MEDICAL AND SURGICAL REPORT OF BELLEVUE AND ALLIED HOSPITALS IN THE CITY OF NEW YORK

VOLUME I

1904

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Compliments of

S. T. Armstrong, M. D.,

General Medical Superintendent,

Department Bellevue and Allied Hospitals.

MEDICAL AND SURGICAL
REPORT
OF
Bellevue and Allied Hospitals
IN
THE CITY OF NEW YORK.

VOLUME I,

1904.

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THE BELLEVUE HOSPITAL SYSTEM OF TAKING AND PRESERVING HISTORIES OF CASES.

DEVISED BY THE COMMITTEE ON CLINICAL RECORDS.

Robert J. Carlisle, M. D.; Warren Coleman, M. D.; Thomas A. Smith, M. D., and Edmund L. Dow, M. D.

Any system of filing histories of cases in a large hospital is of value to the extent to which it permits easy access to the files and economizes space.

The large number of cases treated at Bellevue and the Allied Hospitals makes the question of taking and preserving histories a subject of much importance and great complexity. The number of discharges and deaths in these hospitals for the years 1901, 1902 and 1903 were as follows:

HOSPITAL.	1901.	1902.	1903.
Bellevue	23,491	23,048	27,463
Gouverneur	3,336	2,969	3,946
Harlem	3,314	2,332	2,944
Fordham	1,140	1,110	992
Total	31,281	29,459	35,345

These figures show the need of elasticity in any system to be adopted in these hospitals. Because of the fact that the Bellevue Out-Patient Department was closed several months for repairs the number of outdoor patients is not included in this list.

The system herein described, which may be designated the Bellevue Hospital System, was installed by the Board of Trustees and the Executive Committee on January 1, 1904, and since then has been found to meet all requirements.

The history requirements of the two large classes of patients, the indoor and the outdoor, necessarily differ materially. An out-patient may appear but once to be treated, yet from the standpoint of hospital statistics, both sociological and clinical, it is quite as important to have records of these patients as of the bed patients in the hospital proper.

NOMENCLATURE.

A prerequisite to filing histories under the names of diseases, which is the basis of this system, instead of under the names of patients or by serial numbers, is absolute uniformity in the terms employed in diagnosis. In accordance with such demand, the Committee on Clinical Records compiled* "A Nomenclature of Medical and Surgical Diseases and Conditions," which, upon its adoption by the Board of Trustees, was printed for use by the Visiting and House Staffs of Bellevue and Allied Hospitals. This nomenclature is followed in both the indoor and outdoor departments, in order that there may be no conflict of terms when cases with their histories are sent from the Out-Patient Department to the General Hospital.

The nomenclature is divided broadly into four parts, viz.: Medical, Surgical, Gynecological and Obstetrical. The main heads of the Medical division are:

Diseases of the Blood.
Diseases of the Circulatory System.
Diseases of the Digestive System.
Diseases of the Ductless Glands.
Infective Diseases.
Diseases of the Kidneys.
Diseases of the Lymphatic System.
Miscellaneous Diseases and Conditions.
Diseases of the Nervous System.
Parasitic Diseases and Parasites.
Poisonings and Intoxications.
Diseases of the Respiratory System.
Diseases of the Spleen.
Diseases of the Mind.

The main heads of the Surgical division are:

Diseases of Bone.

---

*The committee received invaluable assistance in compiling the Nomenclature from Drs. William E. Studdiford and William C. Lusk.

Injuries of Bone.
Diseases of Bursæ.
Injuries of Bursæ.
Diseases of the Circulatory System.
Injuries of the Circulatory System.
Congenital Malformations.
Diseases of the Digestive System.
Injuries of the Digestive System.
Diseases of the Ear.
Injuries of the Ear.
Diseases of the Eye.
Injuries of the Eye.
Gangrene.
Diseases of the Genito-urinary System.
Injuries of the Genito-urinary System.
Herniæ.
Infective Diseases.
Injuries.
Diseases of Isolated Glands.
Injuries of Isolated Glands.
Diseases of Joints.
Injuries of Joints.
Diseases of the Lymphatics.
Injuries of the Lymphatics.
Miscellaneous Diseases and Conditions.
Diseases of the Muscular System.
Injuries of the Muscular System.
Diseases of the Nervous System.
Injuries of the Nervous System.
Diseases of the Respiratory System.
Injuries of the Respiratory System.
Diseases of the Skin, Hair and Nails.
Tumors.

The main Heads of the Gynecological division are:
Diseases of the Ligaments, Tubes and Ovaries.
Injuries of the Pelvic Floor.
Disorders of Menstruation.
Diseases of the Urethra.
Diseases of the Uterus.
Diseases of the Vagina.
Diseases of the Vulva.

Following the above is the Obstetrical division.

One of the principal features of the Nomenclature is its alphabetical arrangement. This has been followed almost without exception. The advantage of this arrangement lies in its elasticity as concerns the addition of new terms without disturbing the whole. No deviation, however, from a term accepted for diagnostic purposes is permitted. These terms have been made as comprehensive as possible in order to reduce their number. They are the only ones allowed on the bedside cards and the only ones employed in compiling statistical tables. When amplification of a diagnosis is necessary it may be made upon the history according to directions given in the Nomenclature. For example, the term Chronic Cardiac Valvular Disease is employed upon the bedside card for all cases, not infective, of old-standing lesions of the heart valves, while upon the history this diagnosis is amplified by a statement of the valve or valves affected and the nature of the lesion. For statistical purposes simply a statement of the number of cases of chronic cardiac valvular disease is all that is needed, while such other facts as may at any time be desired will be found upon the histories.

The completed histories are filed according to the alphabetical arrangement of the Nomenclature, and are therefore self-indexing.

Since the methods of disposing of the histories of the indoor and outdoor patients differ so materially they will be described separately.

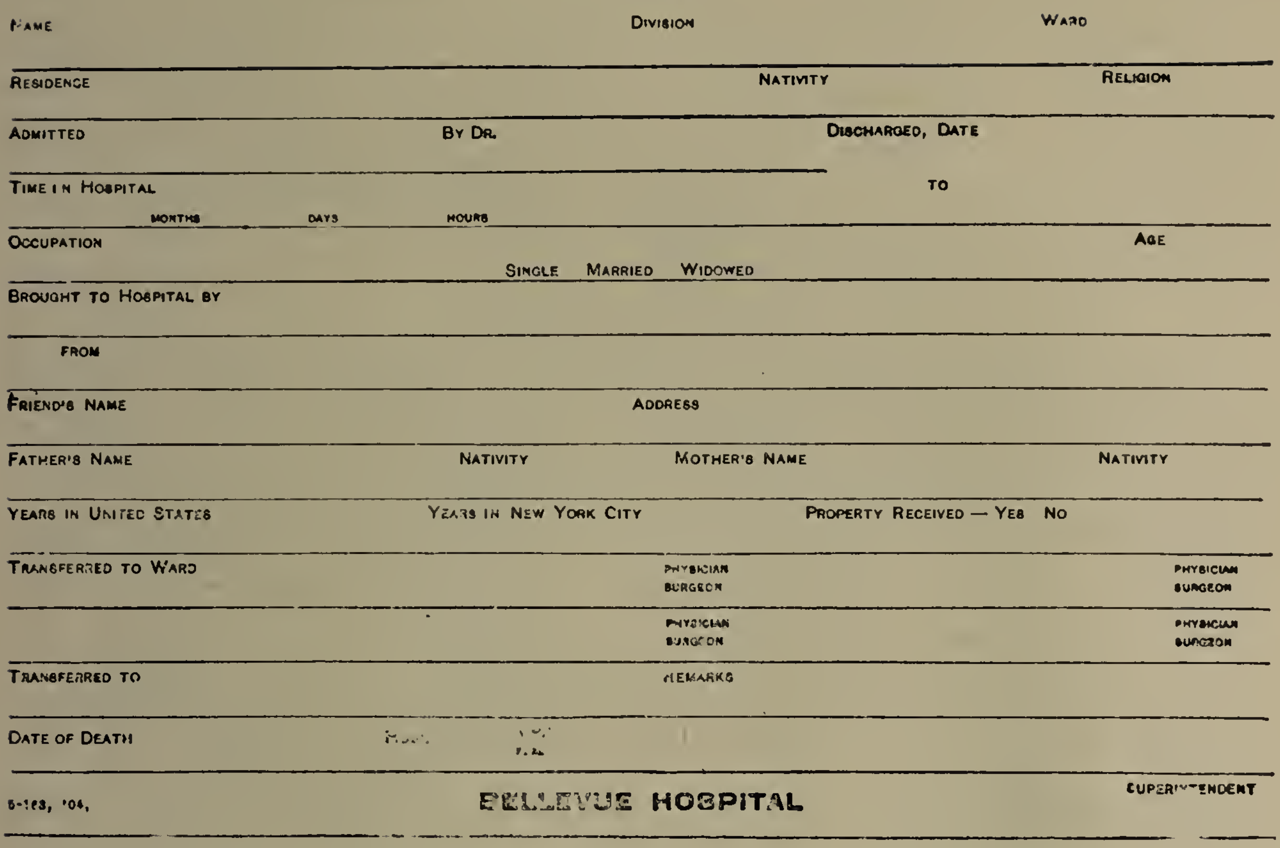
Indoor Service.

THE BEDSIDE CARD.

On the admission of a patient to the hospital a card is filled out in the Admission Office, which card accompanies the patient to the ward and remains at the bedside until the completion of the case. This is the bedside card. It is a stock card such as is used by libraries for card-index purposes, 6 by 4 inches in size. The face of the card when filled out contains all the sociological and other data concerning the patient as an inmate of the hospital, while the back of the card is reserved for the facts which pertain to the patient as a case of disease.

A facsimile is given below of each side of the card.

Obverse.



Name Division Ward

Residence Nativity Religion

Admitted By Dr. Discharged, Date

Time in Hospital to

Months Days Hours

Occupation Age

Single Married Widowed

Brought to Hospital by

From

Friend's Name Address

Father's Name Nativity Mother's Name Nativity

Years in United States Years in New York City Property Received — Yes No

Transferred to Ward Physician Surgeon Physician Surgeon

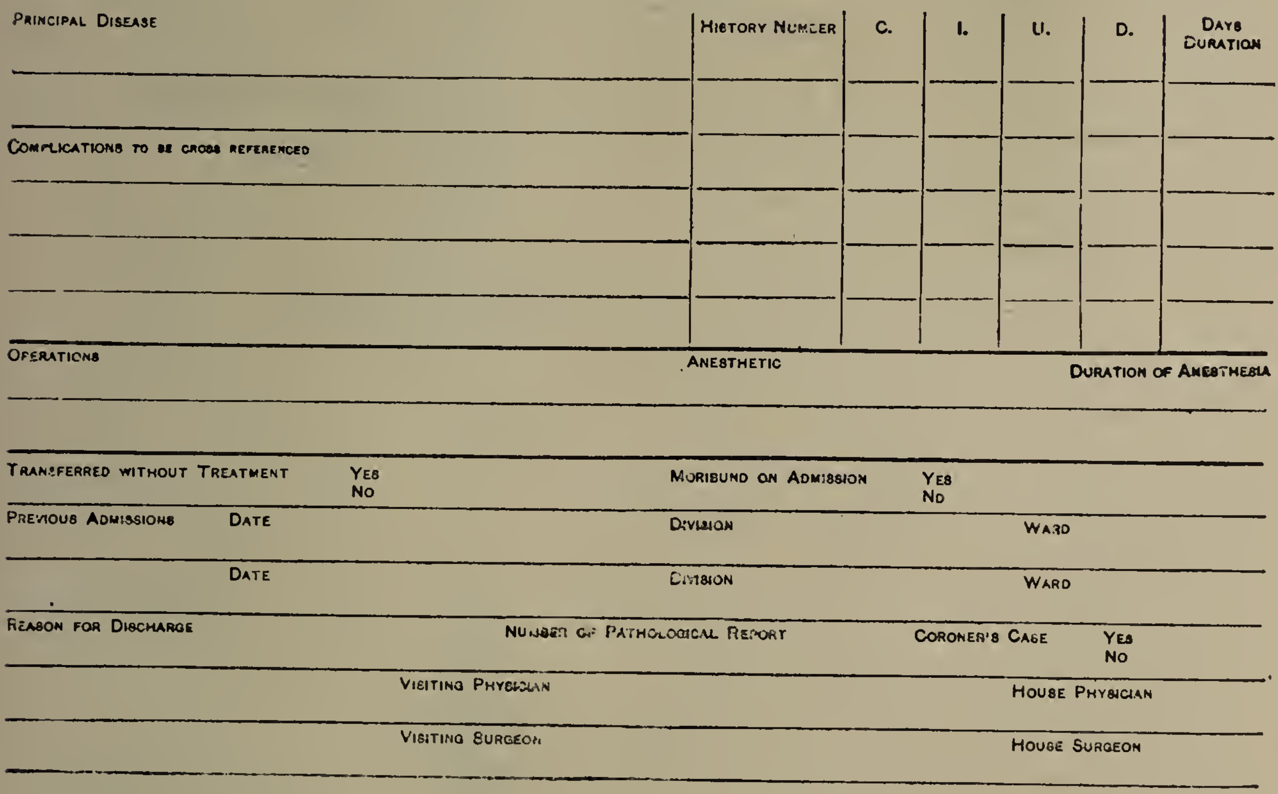
Physician Surgeon Physician Surgeon

Transferred to Remarks

Date of Death Hour A.M. P.M.

6-163, '04, **BELLEVUE HOSPITAL** Superintendent

Reverse.



Principal Disease	History Number	C.	I.	U.	D.	Days Duration
Complications to be cross referenced						

Operations Anesthetic Duration of Anesthesia

Transferred without Treatment Yes No Moribund on Admission Yes No

Previous Admissions Date Division Ward

Date Division Ward

Reason for Discharge Number of Pathological Report Coroner's Case Yes No

Visiting Physician House Physician

Visiting Surgeon House Surgeon

Immediately on the discharge or death of a patient the diagnosis is properly written out in strict accordance with the nomenclature on the reverse side of the card by the house physician or house surgeon, and the card is delivered by the nurse in charge to the administration office. Each day the entire number of cards representing the discharges and deaths of the previous twenty-four hours are delivered by the administration office to the record room. There they are at once put temporarily in a drawer which is divided into compartments to represent the several divisions of the hospital service, medical, surgical, etc.

The completed histories are delivered to the record clerk by the house staff. The clerk thus has a check upon the delivery of the histories in the bedside cards. It is to be noted that no account is taken of a patient by the record room until the completion of the case either by discharge or death.

HISTORY BLANKS.

The histories comprise the original records made by the members of the house staff and nurses on blank forms similar to those used in most hospitals. Because of the necessity of economizing space in dealing with so great a number of histories these forms are printed on as large-sized sheets as is thought to be consistent with east of handling. This size is 14½ inches by 10½ inches, 1½ inches being left blank on the left margin for binding, and ¾ inch on the right margin for trimming. For the same reason the paper used is thin bond, all being white paper except the Medication Sheet and the Post Mortem Record Sheet, which are blue and buff, respectively, because these are always the last sheets in the completed history, and thus by the color in the bound volume allow of easy distinction being made between individual histories. The blank forms are the following:

Medical History Sheet.
Surgical History Sheet.
Continuation of History and Bedside Notes.
Physical Examination Sheet.
Clinical Pathology Sheet.
Medical Temperature Chart.
Surgical Temperature Chart.
Medication Chart.
Ante-Partum Chart.
New-Born Child Chart.
Post Mortem Record Sheet.

All these forms are printed in copying ink, and all writing is done in copying ink and only upon one side of the sheet. This is to provide for a copying process to be described below. Illustrations of these various sheets follow. On the Medical and Surgical History Sheets the items to be inquired into respecting the family history, previous personal history and existing illness or injury are printed near the top of the page just underneath the main heading of the sheet, the sheet below being a plain blank sheet not divided by columns or other device.

MEDICAL HISTORY SHEET.

LEAVE BLANK FOR BINDING.

BELLEVUE HOSPITAL.

SERIAL No.______

NAME______________ M. S. W. NATIVITY______________ MEDICAL DIVISION, WARD______

OCCUPATION__________ AGE______ ADMITTED__________ 190____ DISCHARGED__________ 190____

DIAGNOSIS______________________ COMPLICATION______________________

VISITING PHYSICIAN______________________ HOUSE PHYSICIAN______________________

FAMILY HISTORY. Gout, Rheumatism, Syphilis, Tuberculosis, Alcoholism, Nervous Affections, Cancer, and Family Deaths.	PREVIOUS HISTORY. Habits, Occupation, Diseases of Childhood, Venereal History, Rheumatism, Malaria, Typhoid, etc., Previous Similar Attacks, Change in Weight.	PRESENT ILLNESS. Onset and Chief Complaint, Nervous Symptoms, Alimentary Symptoms, Abdominal Symptoms, Respiratory Symptoms, Cardiac Symptoms, Skin, Joints, Muscles, Genital Organs, Kidneys.

This blank is 10½ x 14½ inches, with 45 ruled lines.

LEAVE BLANK FOR BINDING.

SURGICAL HISTORY SHEET.

Leave Blank for Binding.

BELLEVUE HOSPITAL.

SERIAL No.______

Name____________ M. S. W. Nativity____________ Surgical Division, Ward______

Occupation____________ Age______ Admitted______ 190___ Discharged______ 190___

Diagnosis____________ Complication____________

Visiting Surgeon____________ House Surgeon____________

FAMILY HISTORY.	PREVIOUS HISTORY.	PRESENT ILLNESS OR INJURY.
Gout, Rheumatism, Syphilis, Tuberculosis, Alcoholism, Nervous Affections, Cancer, and Family Deaths.	Habits, Occupation, Diseases of Childhood, Veneral History, Rheumatism, Gout, Tuberculosis, Malaria, Typhoid, etc.; Previous Similar Attacks, Previous Injury (Dates), Complications and Sequels.	*Disease:* Time and Mode of Attack; *Injury:* Time, Part or Parts, Mode of Production, Symptoms (Shock, Deformity, Impairment or Loss of Function, Hemorrhage), Ability to Walk. *Other Abnormal Condition:* Time and Manner of Occurrence, Course, Treatment Previous to Admission.

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CONTINUATION SHEET.

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CONTINUATION OF HISTORY
AND
BEDSIDE NOTES

Name __________ Admitted __________ 190 ____ Ward __________

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PHYSICAL EXAMINATION SHEET.

LEAVE BLANK FOR BINDING.

PHYSICAL EXAMINATION.

NAME________________ **ADMITTED**________________**190**______ **WARD**________

General Appearance, Eyes, Mucous Membranes, Tongue, Throat, Heart, Arteries. Pulse,	Lungs, Abdomen, Liver, Spleen,	Kidneys, Skin, Bones, Joints, Muscles,	Lymph Nodes, Reflexes, Sexual Organs.

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CLINICAL PATHOLOGY SHEET.

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CLINICAL PATHOLOGY

Serial Number

Name

Division

Ward

URINE.

Physical Characters:—Color; Odor; Transparency; Sediment; Reaction; Specific Gravity.
Chemical Characters:—*a.* Urea; Uric Acid and Urates; Phosphates; Chlorides; Sulphates. *b.* Albumin and Related Bodies; Blood; Hemoglobin and Related Pigments; Bile Acids and Pigments; Sugar; Acetone; Diacetic Acid; *b*-Oxybutyric Acid; Fat; Indican; Drug Changes; Diazo-reaction.
Microscopical Findings:—Number of Epithelial, Blood and Pus Cells; Number, Size and Variety of Casts; Cylindroids; Mucus; Crystals; Amorphous Salts; Bacteria; Animal Parasites.

DATES

BLOOD.

Red Cells:—*a.* Number; *b.* Varieties:—Normal, Microcytes, Macrocytes; Poikilocytes, Normoblasts, Microblasts, Megaloblasts; Anisocytosis.
Hemoglobin:—Percentage; Color Index. **Blood Plates:**
White Cells:—*a.* Number; *b.* Varieties:—Polynuclear Neutrophile, Small Mononuclear, Large Mononuclear; Transitional, Eosinophile, Mast Cells; Myelocytes.
Serum Reaction; Bacteria; Hematozoa:—Tertian, Quartan, Aestivo-Autumnal; **Filariasis; Fibrin; Coagulation Time; Sugar.**

DATES

SPUTUM.

Consistency; Color; Odor; Constituents:—Mucus, Pus, Blood, Epithelium, Lung Tissue, Elastic Tissue, Curschmann's Spirals, Crystals. Bacilli Tuberculosis, Bacilli Influenzæ, Diplococci, Streptococci, Staphylococci, etc.

DATES

GASTRIC CONTENTS.

Quantity; Color; Water; Mucus; Bile; Blood; Pus; Mucous Membrane; Proteid; Fat and Carbohydrate Residue;
Odor; Reactions: Free and Combined Hydrochloric Acid; Total Acidity; Acetic, Lactic and Butyric Acids; Enzymes and Zymogens.
Microscopical Examination:—Boas-Oppler Bacillus, Etc.

DATES

FECES.

Consistency; Color; Odor; Reaction; Constituents:—Muscle Fibres and Cell Nuclei; Connective Tissue Fibres; Fat Droplets and Fatty Crystals; Starch Cells; Milk Coagula; Other Food Elements; Epithelial Cells; Membrane; Other Crystals; Mucus; Pus; Blood; Bile Pigment; Coloring Matter; Bacteria; Animal Parasites.

DATES

DISCHARGES AND EXUDATES.

DATES

Leave Blank for Binding.

MEDICAL TEMPERATURE CHART.

LEAVE BLANK FOR BINDING.

Name.................... **Admitted**.................... **19**........ **Division, Ward**........ **Temperature Chart No.**..........

Date		**111°**
Day of Disease		**110°**
Hour		**109°**
108°		**108°**
107°		**107°**
106°		**106°**
105°		**105°**
104°		**104°**
103°		**103°**
102°		**102°**
101°		**101°**
100°		**100°**
99°		**99°**
98°		**98°**
97°		**97°**
		96°
Pulse		**95°**
Resp.		**94°**
Total Urine		**93°**
Total Def.		**92°**
		91°

Diagnosis....................

Serial No.....................

LEAVE BLANK FOR BINDING.

SURGICAL TEMPERATURE CHART.

LEAVE BLANK FOR BINDING.

Diagnosis............................ Serial Number............................

Name.............................. Admitted.............................. 19...... Division.............. Ward..............

Date	Day of Disease	Hour	107°	106°	105°	104°	103°	102°	101°	100°	99°	98°	97°	Pulse	Respiration	Ounces Urine	Defecations	Nourishment	Leucocytes			BEDSIDE NOTES.

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ANTEPARTUM CHART.

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BELLEVUE HOSPITAL

ANTEPARTUM EXAMINATION

History Number

Name **Age** **Nativity** **Occupation**

Address **Division**

Married ; Unmarried ; Widowed.

Admitted 190 **Para........** **Date of Expected Labor**

Date of Examination 190 **by** **M. D. ;** **M. D.**

Age at first menstruation Character menstruation : type Date of last menstruation
pain
amount

Medical History :

Syphilis ; Cardiac disease Kidney disease Diseases of childhood

Age when first walked ;

Previous Pregnancies :

Number at full term Number interrupted Number of abortions Complications

Previous Labors :

Instruments used in Number of still-births Character :—

Previous Puerperia :

Fever present in Abdominal pain Inflammation of breasts Days in bed

Examination : Heart Lungs Breasts Nipple Gland

Urine

Fetal heart-rate Fetal movements Fetal presentation Position

Measurements in centimetres : Pubes to ensiform Pubes to fundus Pelvic circumference

Spines Crests External conjugate Right oblique Left oblique Height symphysis Pubic arch

Pelvic floor Diagonal conjugate Estimated true conjugate Pelvic inclination Weight Height

Vaginal secretion Cervix

Remarks : Abnormalities

Labor : True pains began at M. 190 Temperature Pulse

FIRST STAGE

Fetal heart-rate Frequency uterine contractions Character

Cervix character Membranes { intact / ruptured Presentation Position

Displacements : Head Cord Arms Legs Placenta

Hemorrhage Medication Chloroform

Remarks :

SECOND STAGE

Fetal heart-rate Cervix fully dilated at M. Membranes ruptured { Spontaneously / Artificially } at M.

Presentation Position Displacements : Head Cord Arms Legs Placenta

Hemorrhage Medication Chloroform

Child born at M. 190 Cord wound around neck body extremities

Remarks :

THIRD STAGE

Placenta delivered { Spontaneously / Artificially } at M.

Hemorrhage Pelvic floor Cervix

Temperature one hour after labor Pulse Medication

Remarks :

Summary : Duration of First Stage : Hours Minutes

Duration of Second Stage : Hours Minutes

Duration of Third Stage : Hours Minutes

Duration of Entire Labor : Hours Minutes

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NEW-BORN CHILD CHART.

RECORD OF NEW-BORN CHILD.

SERIAL NUMBER

BORN M. 190 RECORD NUMBER OF MOTHER.

Name Name of Mother Sex of Child

Duration of gestation months. Duration of Labor hours minutes. Character

Presentation Position Character heart sounds at birth Primary respirations

PLACENTA.	DIAMETERS	CM.	CIRCUMFERENCES	CM.	WEIGHT OF CHILD		
Shape	Occipito-mental		Occipito-mental		At Birth	lbs.	oz.
Size	Occipito-frontal				1 Day old	lbs.	oz.
Weight	Suboccipito-bregmatic		Suboccipito-bregmatic		2 Days old	lbs.	oz.
Alterations	Fronto-mental				3 Days old	lbs.	oz.
					4 Days old	lbs.	oz.
	Trachelo-bregmatic				5 Days old	lbs.	oz.
CORD :—	Bi-parietal				6 Days old	lbs.	oz.
Length	Bi-temporal				7 Days old	lbs.	oz.
Insertion	Bi-mastoid				8 Days old	lbs.	oz.
Volume	Bis-acromial		Bis-acromial		9 Days old	lbs.	oz.
Peculiarities	Bis-trochanteric		Bis-trochanteric		10 Days old	lbs.	oz.

Total length of child Cm. Vertex-coccygeal length Cm.

GENERAL CONDITION AT BIRTH :—

Living	Stillborn	Macerated	Development	Rectal temperature
Eyes	Breasts	Skin	Nose	Mouth
CRY :—Weak	Absent	Strong	Abnormalities	

DAILY RECORD.

DAY AFTER BIRTH	1	2	3	4	5	6	7	8	9	10
Eyes Nose Mouth Skin Urine Defecations Umbilicus Food Medicine										
Temperature Pulse Respiration										

Cord separated on day. Umbilicus healed on day.

Remarks :

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MEDICATION SHEET.

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MEDICATION CHART

Name Admitted 190 Ward

DATE	OZ. OF URINE	DIET	MEDICATION	NURSES' NOTES

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Leave Blank for Binding.

POST MORTEM RECORD SHEET.

Leave Blank for Binding.

POST=MORTEM RECORD

____________________Pathologist

Name ______________ **Died** ______________**190 Division**_____**Ward**_____

Leave Blank for Binding.

This blank is on buff paper, 10½x14½ inches, with 50 ruled lines.

When the completed history is received by the record clerk the bedside card which corresponds is placed with it, the statement of the diagnosis on the card and history is compared with the nomenclature, the proper serial number of the principal disease is written on the card and on the history, and the card is then placed in another drawer where all cards belonging to the same hospital service are classified alphabetically, in accordance with the nomenclature. Here they remain until the end of the month. It is from this index that the cross-referencing is done and the list of operations and the monthly report are tabulated. When the report is completed the cards are redistributed alphabetically according to the patients' names and put in card-index cabinets, thus forming an index to the files of histories under the names of the patients.

The sheets of the history are assembled together in a definite order and the whole history is then placed in a manilla paper folder, upon the outside of which, along the folded edge, is written the principal disease and the serial number of that disease, the name of the patient and the number of the medical, surgical or gynecological division to which the case belongs. This completed history in its folder is then filed away in the proper cabinet drawer. The cabinets consist of four large superimposed drawers arranged for filing the histories vertically on the edge. Each of these drawers, with the proper guide-cards, will hold about 400 histories. Here the histories are filed alphabetically by the name of the disease in exact correspondence with the order of the nomenclature, which thus becomes an index to the cabinets. All histories of the same disease are thus filed in a group in the order of the discharge from the hospital and are always readily accessible for inspection, and for the making of copies until they are bound. There are then two indices to the files of histories: the Nomenclature, an alphabetical arrangement of diseases, and the Bedside Card Index, an alphabetical arrangement of the patients' names.

THE COPYING PROCESS.

No original history is permitted to be taken under any circumstances from the record room. As an offset to the inconvenience that might arise from a strict enforcement of this rule a copying process has been put in operation whereby fac-simile copies can be furnished when necessary. By this process six legible fac-simile copies of the charts composing the history can be made in as many minutes. The copying is done by a copying machine such as is used in many business houses.

For the proper carrying out of this process all the blank forms are printed in copying ink; no ink but copying ink is used in writing the histories and all writing is on one side of the sheet only. It has been found that the best two inks for making a good contrast, both before and after copying, are black and red copying ink; combined copying and writing fluid will not give as good a copy as a plain copying ink. The ink is allowed to dry; no blotters are used. The interval of time that may be allowed to elapse before the copy is made has not been determined, but copies have been made seven months after the original was written which differ in no way from copies freshly made. After the histories are bound, copies can be made by the ordinary letter press method.

CROSS REFERENCING.

For this purpose two blank forms are used. The first of these forms is called the Complications Sheet, the illustration of which follows:

COMPLICATION SHEET.

Division.........................

COMPLICATIONS

OF

....

Dates

From...19

To...19

	Names of Complications	History Numbers of the disease named above:	

This blank is 10½ x 14½ inches, with 63 ruled lines.

All the complications that occur in the histories of one disease are named in the column on the left of the sheet. Opposite each complication is written the serial number or numbers of those histories of the disease named at the top of the sheet which show such complication.

The other blank form is called the Cross Reference Sheet, the illustration of which follows:

CROSS REFERENCE SHEET.

DIVISION .... DATES

CROSS REFERENCE SHEET

From 19

NAME OF COMPLICATING DISEASE .... To 19

	Names of Diseases Complicated :	History Numbers of the Diseases named below :	

This blank is 10½ x 14½ inches, with 63 ruled lines.

All the principal diseases, the histories of which are included in other volumes, and which there describe as a complication the disease noted at the top of this sheet, are named in a left-hand column, with the serial number of their histories written opposite.

Both these forms are printed on ledger paper and are of the same size as the history blanks. Each sheet—the Complications and the Cross Reference Sheet—is put in a manila folder and filed with the appropriate group of histories in the cabinet, and later bound with them. For example, given a case of lobar pneumonia complicated by suppurative pleurisy; on the *Complications Sheet,* which is filed at the head of the group of histories of Lobar Pneumonia cases, in the left-hand column of the sheets, is written *Suppurative Pleurisy*, and opposite it the serial number of the history in the Lobar Pneumonia group, which shows this complication, say, 32; while on the *Cross Reference Sheet* which is filed at the head of the group of histories of Suppurative Pleurisy cases, in the left-hand column of the sheet, will be written *Lobar Pneumonia,* and opposite this the serial number of the same history in the Lobar Pneumonia group, *i. e.*, 32. This is very simple in practice, and one can see by a glance at these sheets all the complications of which there are records in any group of diseases, and also, on the other hand, all the cases filed elsewhere in which any one disease occurs as a complication. These Complications and Cross Reference Sheets are written up by the record clerk from the bedside cards, as is stated above.

The proper relationship of two concurrent diseases is often perplexing. As uniformity in this regard is necessary for the proper tabulation of principal diseases and complications, the following directions were formulated, based in the main on the directions given in the circular issued by the United States Census Office, entitled, International Classification of Causes of Death:

"For the purpose of obtaining uniformity throughout the hospital in regard to the manner of stating diagnoses on the bedside cards and the histories, it is ordered by the Executive Committee that the following rules be adhered to in stating the relation between the principal disease and the complication or concurrent disease:

"1. When the disease for which the patient enters the hospital is *dependent upon another co-existing disease,* the latter should be named as the principal disease and the former as the complication; *e. g.,* Chronic Cardiac Valvular Disease and

Chronic Cardiac Dilatation; the reverse is often written; Chronic Pulmonary Tuberculosis and Serofibrinous Pleurisy, and not the reverse; Chronic Morphine Poisoning and Colitis.

" 2. When a *surgical condition complicates a medical case* it should be named as the complication and not (as is usually done) as the principal disease when the diagnosis is completed on the Surgical Division, *e. g.,* Diabetes Mellitus and Gangrene; Lobar Pneumonia and Suppurative Pleurisy, and vice versa when a *medical condition complicates a surgical case* it should be named as the complication and not (as is usually done) as the principal disease when the diagnosis is completed on the Medical Division, *e. g.,* Fracture of Femur and Alcoholic Delirium, and not the reverse.

" 3. Of two *independent* affections the choice should be made in favor of—

> " 1st. An infective disease, *e. g.,* Lobar Pneumonia and Atrophic Cirrhosis of Liver.
>
> " 2d. The graver affection, *e. g.,* Carcinoma of Uterus and Nephroptosis.
>
> " 3d. The disease for which the patient applies for treatment, *e. g.,* Zoster and Acute Rhinitis."

FINAL DISPOSITION OF THE HISTORIES.

When, in the opinion of the Committee on Clinical Records, a sufficient number of histories have accumulated to give a volume of suitable size, taking into consideration also the needs of the copying process, the histories are taken from the cabinet and bound in volumes in the same order in which they are filed serially numbered in each group with the proper Complications and Cross Reference Sheets in the fore part of each volume.

The bound volumes, distinguished by dates and the serial numbers of the histories included therein, are shelved according to the same alphabetical arrangement followed in the Nomenclature and in the filing cabinets; the Nomenclature forming an index to the groups of diseases both of the bound volumes and the cabinet files.

II.—OUTDOOR SERVICE.

The histories of patients applying for treatment in the Out-patient Department are recorded upon folded sheets of paper and filed by the card-index system. This causes them to be more valuable and accessible than if they were written seriatim in bound volumes. A sheet of paper is preferred to a sheet of cardboard, because the question of the space to be occupied by over one hundred and thirty thousand histories each year is materially important. It is preferable to individual books, as these are too large for patients who come to the dispensary but once, and are not extensible should they become filled with data. If broad enough to provide for clinical notes and prescriptions side by side, individual books are too clumsy. Inasmuch as the history should not be limited in extent by the space afforded, double sheets of paper are folded so that they may serve as covers to single sheets, which can be inserted as required. The folded edge of each sheet moreover permits an easy separation of the histories when they are pressed together and filed vertically upon the opposite edges in a drawer. An illustration of this sheet is given herewith; the size of the page is 9½ by 12 inches:

OUT-PATIENT DEPARTMENT HISTORY SHEET.

BELLEVUE HOSPITAL

OUT-PATIENT DEPARTMENT

Race *Age* *M. S. W.* *Male*
Female

Occupation :

Family History :

Previous History :

Present Illness :

Physical Examination :

Division

Name

Date

Address

Record Number of Hospital History

Primary Diagnosis :

Date	Clinical Notes	Treatment	Blank

Histories are filed by the card-index system. They may be arranged and rearranged according to any method desired, removed whenever required, and replaced with the least possible difficulty.

Several essential features are prominent in this system.

The histories are complete records of the sociological data, the medical history, the primary diagnosis, the entire treatment, its subsequent results from day to day and the final diagnosis.

Separate prescription blanks are not permitted.

The histories are filed temporarily by the names of the patients and eventually by the names of the diseases treated.

Each department has its own file of histories.

The history accompanies the patient when he is sent to the wards of the hospital.

The practical working of this system has proved it to be extremely simple and easy to maintain.

When a patient enters the Out-patient Department for the first time he goes to a desk where his name, address, occupation, age, nationality and other desired sociological data are entered upon a folded history sheet. The date and the division to which he is to be assigned are then also stamped upon this sheet. All of these data are taken by a paid clerk, thus relieving the physicians of this purely clerical work. The name, address, date and division are put on the left border of the folded sheet so that they will be uppermost when the history is filed on its right edge. This history sheet is then put in a plain manilla envelope, which serves the double purpose of keeping the history sheet clean and of making difficult its inspection by the patient.

At this same time the patient receives from the clerk, for permanent preservation, a card bearing the patient's name and address, the division to which he was assigned, the date and the hour at which he should return for further treatment. This work is facilitated by employing the stamps used upon the corresponding history sheet. These cards serve to identify the patient when he applies at the Out-patient Deparment for treatment at a later date.

The patient retains this card and carries the envelope containing the history sheet with him to the benches, where he awaits his turn for the room or division to which he has been assigned. His place in line is secured by a small colored card bearing the appropriate number of succession. By noting how many of these cards of each color have been given out to both those reapplying for treatment and those applying for the first time, the desk clerk at the close of the day is able to record how many patients went to each department. In addition to these facts the clerk also tabulates in a book provided for the purpose any desired sociological data at the same time that they are noted upon the individual history sheets.

When a patient is called for treatment into one of the rooms the physician records upon the history sheet the history of the patient's family and of the patient's life which is pertinent, and a statement of his former diseases and his present illness. This is followed by a record of his physical examination and any important laboratory evidence. The physician then writes across the left margin of the sheet the diagnosis by which the history is eventually to be filed when he can do so satisfactorily; otherwise a provisional diagnosis is written there, and the correct diagnosis is inserted when the history of the case is completed at a later date.

The second and subsequent pages of the history sheet are each divided into four columns. The first is for the dates of subsequent treatments; the second for clinical notes recorded each time the patient returns to the Out-patient Department, and for treatment administered by the physician himself. The third column is for the prescriptions ordered from the apothecary for the patient. The narrow fourth column is for mucilage, should extra single sheets be inserted.

After the physician has recorded the patient's history, examination and provisional diagnosis, he writes the prescriptions desired in the column mentioned for that purpose, puts the history sheet thus completed in a manilla paper envelope and hands it to the patient, who takes it to the apothecary's window.

The apothecary takes the history sheet from the envelope and numbers each new prescription there inscribed with a colored pencil seriatim, so that each prescription may subsequently be ordered by number if desired. The blank manilla envelopes are put to one side to be used by clerk or physician on a subsequent day.

The medicines are dispensed to the patient in containers having labels bearing the patient's name and the directions for taking. The name of the patient is obtained from the history sheet upon which the prescription was written. Its presence upon the label prevents a patient receiving medicine intended for another.

At the end of the day the apothecary files these history sheets upon their edges in drawers, arranging them alphabetically according to the names of the patients, facilitated by two hundred and fifty guide-cards in each drawer.

At Bellevue Hospital Out-patient Department there are four medical, four surgical, four gynecological, four children, four genito-urinary and four tuberculosis divisions. Cabinets of four drawers each are kept in the various rooms. Each division has a drawer in a cabinet in which its histories are thus filed at the end of the day by the apothecary. These drawers are frequently removed from the cabinets and placed upon the tables when they are in use.

If a patient applies a few days later for further examination and treatment he enters the room in turn and presents his identification card to the physician. The physician finds the former history and record of treatment which has been filed in his drawer alphabetically by the name of the patient. Should there be two or more histories bearing the same name the address written beneath the name will differentiate them. If the patient has forgotten or lost his identification card the history may be found as easily by having the patient give his name. It is believed that the ease of finding the history when a patient has lost his card makes a system of filing histories by name rather than by an arbitrary number the most convenient one. Inasmuch as the prescriptions sent to the apothecary are written only on the history sheet it follows that unless the original history is found it will not be possible to prescribe for the patient unless the patient applies at the clerk's desk for a new history sheet with its data.

The advantage of compelling the physician to have recourse to the original history each time the patient applies for medicine is very great. Then there will be no excuse for the omission of clinical notes which are pertinent to the case and which should be recorded with the prescriptions and opposite to them.

Moreover, in any system where prescriptions are ordered from the apothecary on separate sheets of paper or special prescription books, the physician must copy the prescription again on the history if the record is to be complete, and this extra work imposed upon the physicians, may, and often does, lead to incomplete records.

A simple system that compels completeness is the one most to be desired.

The physician having obtained from the drawer the original history, records the result of the former treatment and in the adjacent column writes the prescriptions to be now obtained from the apothecary. The history is put in a manilla envelope and handed to the patient who follows the same procedure as before.

Should the patient be sent to a ward of the hospital the messenger who accompanies him takes the history to the admission office and then to the ward with the patient, where it is filed at the bedside with the ward history. The house staff is thus accurately informed what information the Out-patient Department possesses and what treatment the patient received. It is apparent that the advantage to them of having this history contain the complete record of prescriptions ordered is considerable. If the patient is not very sick and prefers to go home and see his family before he enters the hospital, he is told to apply to the Out-patient Department when he returns for admission, and then the procedure mentioned is followed.

When the patient leaves the ward of the hospital and his ward history is sent to the hospital record room, this Out-patient Department history accompanies it. The record clerk then writes upon it the diagnosis written upon the ward history and the number of that history, so that this corresponding ward history may be referred to by the Out-patient Department physician. The Out-patient Department history is then sent by the record clerk to the apothecary in the Out-patient Department, who files it in the drawer where it belongs according to the plan before mentioned, where it is ready for the patient when he again applies for treatment during his convalescence.

An epitome of the ward history is recorded in the column provided for clinical notes by the ward physician.

Moreover the reference provided by the record clerk to the easily accessible history makes the Out-patient Department history complete.

There remains to be described the method by which these histories are eventually preserved so that they shall be useful and available for medical purposes and at all times accessible as individual records. If it is desirable that physicians should make use of these histories by studying a group of cases or by collecting and comparing any of the data they may have recorded, then a system should be used which will facilitate this work. The system should not only allow the desired group of histories to be obtained easily, but more especially should permit them to be replaced in their proper positions without tiresome labor; otherwise physicians will seldom avail themselves of the mass of valuable medical data. It is believed that this can only be accomplished by filing and grouping the histories under their diagnoses according to an adopted nomenclature.

At Bellevue Hospital a history is regarded as completed when a patient has not returned for treatment during a period of two months. When a sufficient number of completed histories have collected in a drawer they are removed, and each is inspected to see that the final diagnosis is written upon its left border; otherwise this is then done to agree with the adopted nomenclature. They are then filed as before on their right edges, but in a separate cabinet of drawers for permanent preservation in a room provided for the purpose. In these drawers they are placed according to the arrangement given in the printed nomenclature already described, and each group is separated by a guide-card bearing the name of the disease or condition. The histories having the same diagnosis are arranged according to the date upon which the patient first applied for treatment. The histories can thus be easily used for statistical or other purposes. Moreover, when this method of filing is employed, a physician may find the history of a certain case when he knows only the diagnosis and perhaps approximately the date of treatment and does not remember the patient's name. However, occasionally a patient returns for treatment one or two years later, and the physician sometimes wishes to obtain the original history, having no information except the name of the patient. This has required that a separate card-index be kept, giving the name of the patient on the upper edge of the card with the diagnosis and date below. These cards are made of very thin cardboard so that they will occupy but little room, are blank, and are filed by the name of the patient. They are filled out by the clerk at the same time the histories are filed by diagnoses.

The fact that only single histories are sought for by name of patient and that groups of histories are utilized by names of disease, is sufficient reason for filing the completed histories according to the latter method for permanent preservation.

This system was adopted by the Board of Trustees before the new Out-patient Department was opened, so that since that time a history is on file for each patient who has applied for treatment to the Out-patient Department at Bellevue Hospital.

A CLINICAL STUDY OF SIXTY CASES OF CEREBRO-SPINAL MENINGITIS.

By Francis Huber, M. D., *Visiting Physician,*

AND

P. W. Monroe, M. D., *House Physician, Gouverneur Hospital.*

It is not our object to enter upon a historical review of the various epidemics which have appeared in this country. J. Lewis Smith to whom we are indebted for one of the best contributions on the subject of Cerebro-Spinal Meningitis, has described the earlier epidemics, including the one which occurred in 1872.

Flexner and Barker have studied the Lonaconing outbreak. In 1898 and 1899 the disease prevailed in a mild form in twenty-seven States. The Boston epidemic was described by Councilman, Mallory and Wright; the one in Baltimore by Osler.

In our report, only the cases admitted to Gouverneur Hospital during March, April and May* of this year will be discussed.

Table of Cases Admitted.

Age.	Under 1 Yr.	1–2½ Yrs.	2½–5 Yrs.	5–10 Yrs.	10–15 Yrs.	15–20 Yrs.	20 Yrs. Or Over.†	Total.
Male	2	1	6	6	1	5	5	26
Female	2	7	8	8	4	5		34
Recovered		1	1	6	2	5	2	17
Died	3	3	7	4	2	3	3	25
In Wards	1	4	5	5	1	2		18

The youngest was about six months, the oldest a man age forty years. Two-thirds of the cases, it appears, occurred in children ten years of age and under. Nearly all were brought in from the large tenement-houses of the lower eastern section. Sturdy, healthy children (the pride of the family or house) were affected more often than the sickly and weak. Of the 25 deaths, 10 occurred within forty-eight hours after admission, as follows: 4 on the first day of disease, 1 each on the second, third and fourth days, and 3 on the fifth day.

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*To my colleague, Dr. F. C. Hollister, sincere thanks are extended for his kindness in permitting me to avail myself of the material and to study the cases admitted to his service during the month of May.

†A brief synopsis of the cases will be found at the end of the article.

Day of Death.

The fatal termination took place on the first day of disease in 4 cases (1 no definite history); second day of disease in 1 case; third day of disease in 1 case; fourth day of disease in 1 case; fifth day of disease in 7 cases; seventh day of disease in 2 cases; ninth day of disease in 1 case; twelfth day of disease in 2 cases; fourteenth day of disease in 2 cases; fifteenth day of disease in 1 case; thirty-second day of disease in 1 case; forty-second day of disease in 1 case; forty-fifth day of disease in 1 case; total, 25.

In looking over the synopsis presented above, we find that 4 died on the first day, 1 each on the second, third and fourth days, and 7 on the fifth day, the period of gravest danger and largest mortality occurring during the first week. In children under five years the prognosis is very grave.

Causes of Death.—In cases living but a few days profound toxæmia caused the fatal issue (usually with convulsions). In one case there was a complicating double pneumonia; in another, an acute nephritis. In several death was preceded by "impairment and perversion of the respiratory centres."

In a man of about twenty-five, chronic alcoholism contributed to a fatal termination. In the prolonged cases death was caused by exhaustion.

Complications.—Conjunctivitis was present in 38 cases. The secretion was muco-purulent in character. Smears were made in 12 cases; an intracellular diplococcus negative to Gram, was found in 9 instances.

Rhinitis occurred in 16 cases. Smears examined in 4 cases showed an intracellular diplococcus negative to Gram, in 2. Herpes was present in 16 patients; it generally made its appearance from the second to the sixth day.

Bronchitis was found in 12; strabismus in 6; pneumonia in 2; stomatitis in 4; nephritis in 2; persisting vomiting in 2 (chronic cases); total deafness in 3; parotitis in 1 (double, subsiding in a few days); cervical adenitis, resolving in a few days, was noticed in 1 patient; hemiplegia occurred once.

Ophthalmic examinations were made in 15 cases by Dr. Martin Cohen. In only 1 instance was a partial optic atrophy O. D. S. discovered.

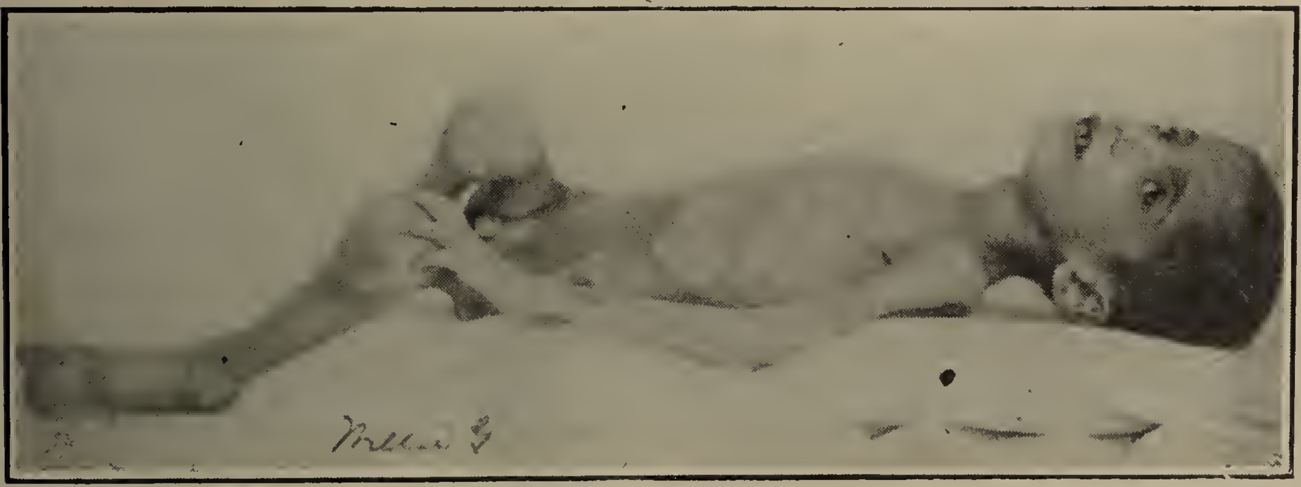
A right-sided facial paraylsis lasting three days was present in 1 case; hydrocephalus occurred in 2; paralysis of the left shoulder muscles in 1 little girl. Arthritis was not noted in any of our cases.

Swelling, redness and tenderness over the dorsum of the hands and wrists, resolving in a few days, was observed once.

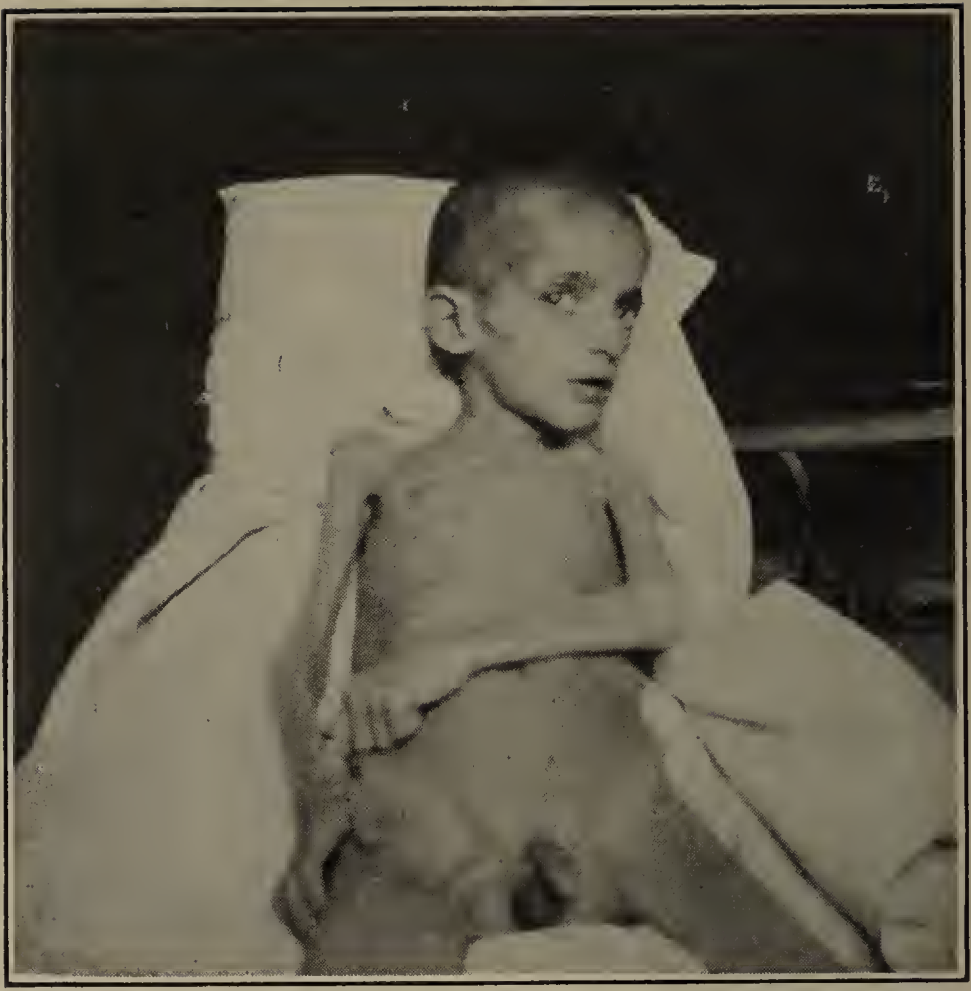
In another, a swelling on the dorsum of the wrist, slightly tender and fluctuating, with considerable redness and tumefaction anterior to the wrist, persisted for several weeks. The anterior swelling gradually subsided, the one over the dorsum persisted, and upon aspiration revealed fluid blood, constituting a hæmatoma.

Sequelæ.—Deafness, blindness, aphasia, mental changes leading to epilepsy, and more or less impairment of the mental faculties, have been noted. Persistent headaches and neuralgic pains may remain.

Chronic hydrocephalus is very apt to develop in a certain proportion in the young, and even in adults. Von Ziemssen gives the following symptom complex as pathogonomic of this sequel: "Paroxysmal and severe headache, pains in the neck and extremities, vomiting, loss of consciousness, convulsions and involuntary discharges of urine and feces." In addition, marked emaciation was present in our cases.



Willie G. Showing the Emaciation and Scaphoid Abdomen. Ill 100 days.



Willie G. 6 years old. Admitted March 15, 1904.
Extreme degree of Emaciation.

Emaciation, at times extreme, is not infrequently observed in cases lasting several weeks or more. Now and then the condition is due to the difficulty in deglutition, loss of appetite and vomiting. In the larger number the loss of flesh, it seemed to us, must be attributed to the trophic disturbances. In the protracted cases we find marked retraction of the abdomen, in addition to the general wasting.

Though infectious, the disease is not regarded as being contagious. It is not at all likely that the disease is communicable from person to person under ordinary circumstances. No instance of ward infection has come under our observation, nor have we been able to find any record of such. Hirsch, Kohlman, J. Lewis Smith and others have reported cases apparently traceable to personal contact. As there is a possibility of this mode of origin, aside from other considerations, all unnecessary intercourse with the affected should be discouraged. In the present series, in four instances, two were ill in the same family (in three, both cases entered the hospital). In another, three children were affected in a family. In two instances two cases were observed in the same house.

In general terms Latimer claims "that the psychical phenomena are most apparent in those cases in which the cortex is most implicated, the special sense disturbances where the base is most involved, and the muscular and general sensory manifestations when the spine is invaded." No such sharply defined clinical states are observed in practice.

In the early stages, "the symptoms are those of exaggerated nerve sensibility, pain, general hyperæsthesia, photophobia, aversion to sound, irregular and forcible muscular contractions, restlessness, delirium, perhaps convulsions, followed by indifference to light, disappearance of pain, the pupils become dilated, and apathy—even coma—may set in."

Indifference, apathy and coma may be present as the result of an intense toxæmia at a very early period, even within a few hours of the onset.

In reviewing our series, we find it difficult to classify a large number of cases (running a very irregular course) under any appropriate heading. For practical purposes Osler's subdivision, in the absence of a better, may be employed. Three forms are described:

1. Malignant;
2. Ordinary;
3. Anomalous forms—
    - (*a*) Abortive type;
    - (*b*) Intermittent type;
    - (*c*) Chronic form, characterized by a series of recurrences of the fever, with an extremely complex symptomatology. The attack may extend over many months and is attended with intense marasmus. The protracted course is attributed to the presence of brain abscesses, or chronic hydrocephalus.

A sudden onset, with rare exceptions, is characteristic of all types. The exact time, to the hour, could be established in many instances. Ordinarily the patient was seized with vomiting, headache, chills or chilly feelings. In infants convulsions were common, in older children chills or "an algid state" were present. Pains in the head, at times in the limbs and back of neck, were observed. The pupils were contracted, now and then irregular. Fever of greater or less intensity was present, with more or less well defined nervous symptoms.

Vomiting during the first day or two was a common occurrence and soon subsided. When this symptom occurs later on in the disease the outlook is grave.

Pain in the head, extending to the neck and back, occurs early and is a constant symptom. It may be frontal, occipital or general. Many children complained bitterly of severe abdominal pains. In other patients shooting pains in the limbs and chest were probably due to a neuritis. In many the spine in particular was very sensitive.

Chills, chilly feelings or an algid stage were frequent initial symptoms. During the first four or five days and even later recurring chills (of no prognostic import) would take place, reminding one of a suppurative process. The microscopic examination of the cerebro-spinal liquor did not bear out this suspicion.

The usual position of the patient in the bed is with the head drawn back, the thighs and legs flexed, with or without a forward arching of the spine. The contraction lasts from two, three to four weeks, and even longer. In some cases a decided amelioration of symptoms would take place, followed by a relapse.

The mental state varies from apathy or indifference to stupor or coma. Acute delirium or restlessness may exist, necessitating restraint at times.

Hyperæsthesia adds to the suffering. The least touch, an attempt to examine, attending to the toilet, etc., causes the patient to resist and cry out.

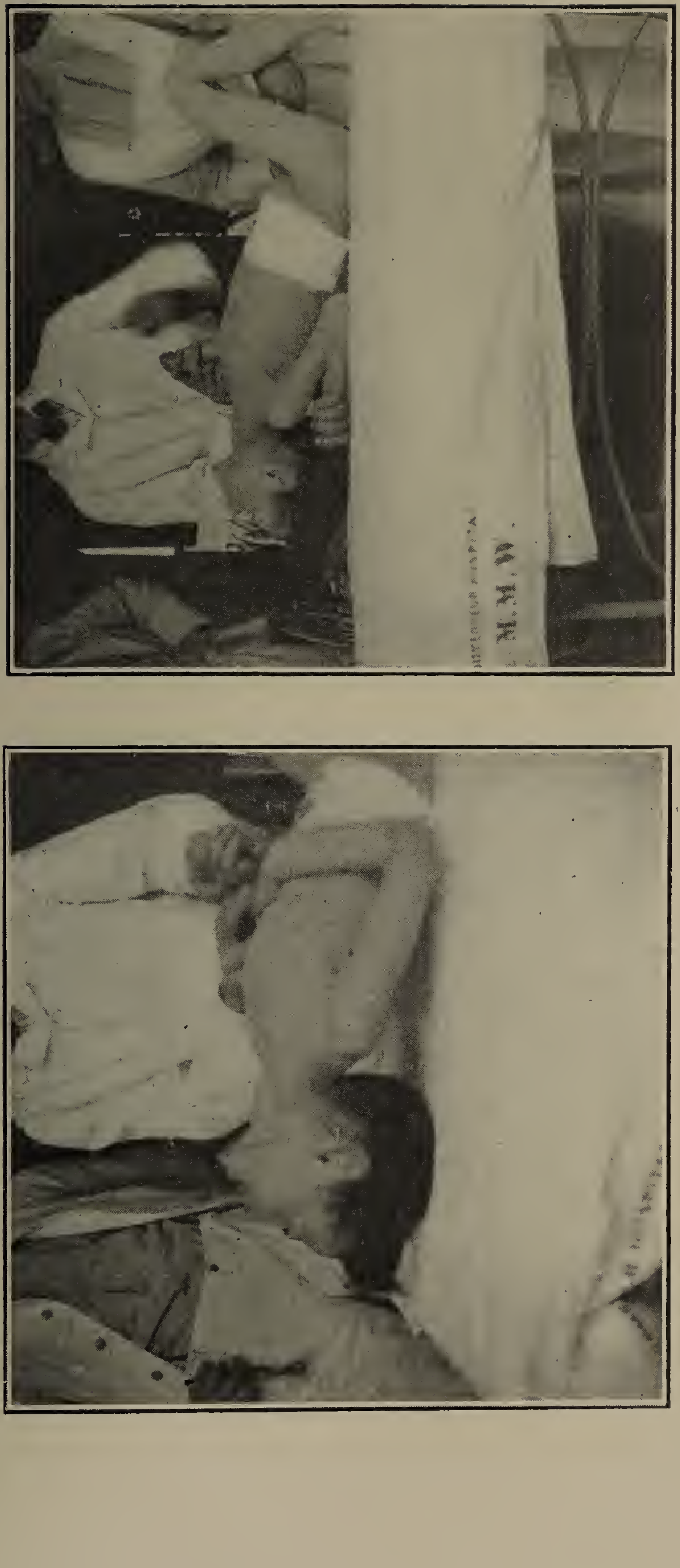
Stiffness and retraction of the posterior cervical and dorsal group of muscles, diagnostic symptoms of great value, occurred early. Rotation was possible, the forward movements only being restricted. Muscular tremors were common; even the muscles of the tongue were involved. Opisthotonos was seen in the severe cases; orthotonos existed in a large number; trismus, however, was observed only in a few.

Genital irritation, evidently of central origin, was an early symptom in many, both in children and adults.

Retention of urine was a frequent complication in the early stages. Suppression or hematuria existed in the malignant type.

Constipation was the rule. Incontinence of urine and feces was a common feature during the stage of coma.

Urinary examinations were made in all cases. Traces of albumin, disappearing in a few days, and occasional casts, were discovered in many specimens. In only two patients were posi-



Retraction of Head.

tive evidences of nephritis present. Hæmaturia, followed by suppression, was met with once. Aside from the increased leucocytosis, the blood did not show any positive changes. Thirty thousand were found on the first day in one instance.

Many of the patients are quiet so long as they are not disturbed. In others the restlessness is extreme, the patients tossing about from place to place.

J. Lewis Smith states that the changes in the pupils, occurring early, consist of dilatation, less frequently contraction, oscillation, inequality of size, feeble response to light, etc. Jacobi tells me that in former epidemics the pupils were usually dilated. In our series contraction was the rule in the earlier stage. The other signs were observed later. Lateral nystagmus was noticed several times.

The temperature does not follow any regular course. Remissions are frequent, the curve is irregular and variable. There may be little or no elevation, with marked retraction, and other evidences of cerebral and spinal involvements.* The pulse, in our experience, in children has usually been rapid; in adults it may be full and strong at first; often, however, it is increased in frequency. The chief characteristic is its irregularity—it may be rapid and feeble or slow and intermittent. Marked variations in frequency during different times in the day and on consecutive days were observed.

No characteristic respiratory difficulty occurs in epidemic cerebro-spinal meningitis. The respiration may be irregular, sighing, or of the Cheyne-Stokes type; in convulsions and coma the peculiarities belonging to those states are noted. General cyanosis is often present. Now and then pulmonary œdema supervenes. In four of the fatal cases death was due to paralysis of respiration. The respirations would become slow, perhaps as low as three per minute, and then stop, the heart continuing to act for a short time longer. Artificial respiration was of no avail under these circumstances.

The reflexes are usually increased during the stage of irritation and diminished later on. Kernig's sign was present at some time in nearly all our cases.

The tongue, teeth and lips were covered with sordes in a fair proportion of cases. Usually there was a heavy coating, which gradually disappeared, leaving a red, raw surface. In a very small number there was but a light fur.

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* Temperature charts illustrating the nervous types will be found at end of paper.

Considerable difficulty in deglutition was met with in many early in the disease. The trouble would be readily overcome when gavage was resorted to a few times.

In a number a history of spots was obtained from the parents. Petechiæ were observed in 14 cases which came in early, the eruption fading within three or four days. In a man of twenty, in addition to the hemorrhagic extravasations, large irregular spots (resembling port wine marks) occurred upon the back and limbs.

The rash was absent in some of the severe and fatal cases; on the other hand, it was quite profuse in some that recovered.

Some of the children presented parallel hemorrhagic streaks upon the abdomen and chest, due to their scratching the integument with their finger nails. Large hemorrhagic extravasations took place in some of the grave cases. Diffuse mottling of the extremities, ears, etc., passing over in a few hours, was common in the early stages; later on, its appearance was of grave prognostic import. The face in the first twenty-four hours was of a pallid hue, the eyes sunken, the surface generally cold.

Transitory irregular patches of erythema upon the face, forehead or ears were frequent. The tache cerebrale could be made out in the routine examinations. Numerous minute red points (resembling the eruption of scarlet) were seen every now and then over the elbows, knees, trochanters and shoulders, particularly marked in restless subjects. Roseola, erythema, nodœsum and urticaria were infrequent occurrences.

No particular difficulty was experienced in distinguishing the drug eruptions due to the coal tar products.

A tendency to hemorrhages in the skin upon slight trauma or at points of irritation was frequent. Strangely, however, no bad results attended the giving of subcutaneous injections of salt solution in these cases. In several prolonged cases purpuric spots occurred late in the disease. In Willie G. we found them on the sixty-first and again on the seventy-first day. In others they persisted until death.

Herpes (once supposed to justify a favorable prognosis) were present in more than one-quarter of the cases. They appeared upon the lips, alæ-nasi, around and upon the ears and cheeks. In one of the chronic cases (Max P.) deep scars remained upon the left cheek.

Prognosis.—If we consider the underlying pathological condition, it is rather a surprise to find that recovery takes place in any well-defined case. The truth of the following statement, made by J. Lewis Smith, has been verified time and again, in our experience, within the past few months: "Cerebro-spinal fever is justly regarded as one of the most dangerous maladies of childhood. It is dreaded not only on account of the great mortality which attends it, but also on account of its protracted course, the suffering which it causes, the possible permanent injury of the important organ which it chiefly involves, and the not infrequent irreparable damage which the eye and ear sustain."

In some of the recoveries the condition was so distressing that death would have been preferable. We have seen children, whom we had every reason to expect would recover, take a turn for the worse; on the other hand, not a few given up by careful and experienced men, unexpectedly recovered. In protracted cases the outlook is not very encouraging; the end is simply deferred, death taking place from exhaustion even as late as eight months after the onset.

The greatest mortality and the most dangerous period are in the first few days, when the intense infection of the meninges, brain and cord produces the marked and characteristic symptoms, with the evidences of profound toxæmia. The younger the child, the greater the danger.

The mortality in this, as in former epidemics, was high. With our large ambulance service many were brought to the hospital in a moribund state and died within a short time of admission. Our cases were not selected; none were refused.

Cases of a malignant type constituted about 10 per cent. of our number. The most rapid case occurred in a well-developed girl of fifteen years, who went to bed at 11 P. M. feeling well. She woke up about 4 A. M. with vomiting and headache, soon became unconscious, and died at 7 P. M. (three hours after admission), fifteen hours from the appearance of the first symptoms.

The duration of the disease varies within wide limits. Death has taken place within five hours; in other instances months may elapse before the fatal issue occurs.

Ordinarily, in cases that run a favorable course, the patient is ill from about two to three weeks. The so-called aborted type runs a stormy but short course, and recovery is assured about the end of the first week. On the other hand, recrudescences or a relapse may prolong convalescence for several months.

Treatment.—The usual general recommendations were followed. Cold to the head, etc., were employed as routine measures. The temperature, when above 103 degrees, was reduced by means of colon irrigations at 80 degrees or mustard packs repeated every three-four or six hours, as required.

Local abstraction of blood was not adopted. Many of our patients had been leeched without much apparent benefit before they were sent to the hospital. Phenacetin, with or without codeine, frequently gave relief to the headache and general pains. In others, codeine or morphia, by mouth or hypodermically, was resorted to to relieve the restlessness and suffering. Iodides, warmly recommended by various authorities, were given as a routine plan.

Fluid diet only was allowed. In the difficult cases forced alimentation (per nasal or stomach tube) was practised. Brandy and whiskey were given freely when the general condition was such as to justify their use.

Various applications have been made to the spine, including Crede ointment, without apparent benefit. In order to relieve the intracranial pressure lumbar puncture was resorted to, with gratifying results for the time being. In a few cases lysol injections were made, with but indifferent results.

Warm baths, T. 95 degrees, given in the later stages, seemed to add to the patient's comfort and quickly relieved the contractures of the extremities and rigid condition generally.

Nursing, "kind, intelligent and efficient," is essential in this disease. I would here extend my thanks and express my high appreciation of the faithful work done by our nurses during this trying period.

Incontinence of urine and feces was a marked feature in our patients. In but one instance, in a boy who had been ill for over two months, a small bedsore developed.

The mortality in the early stage and the inefficiency of the usual treatment led to the early adoption of lumbar puncture as a routine plan. The course of the disease, we must remember, is irregular and variable. The initial symptoms in mild and in severe cases may be as pronounced in the one as they are in the other, and as the subsequent progress is so uncertain, we are scarcely in a position to draw correct conclusions as to the relative value of any plan employed early. It must not be forgotten that not only are the meninges affected, but the brain and cord are also involved; in fact, the disease is properly a meningo-encephalitis and myelitis.

The character of the epidemic, the intensity of the infection and the susceptibility of the patient are important factors, not to speak of the sanitary conditions. Each case must be judged by itself. The unexpected remissions and exacerbations, the apparent establishment of convalescence interrupted by sudden recrudescences, or perhaps relapse, which cannot be foreseen, irregularities characteristic of the course of the malady, leave us in doubt as to the actual success of our therapeutic measures.

In a few instances the improvement was so rapid after admission to the hospital that puncture was not resorted to, though the usual classical symptoms were present. In patients with extreme retraction of the head, marked rigidity of the spine and opisthotonos, a dry tap was the rule in the early stage, though subsequently fluid was obtained in many of these cases.

In resorting to lumbar puncture, an aspirating needle of small calibre (½ mm. in diameter), 2½ to 4 inches long. was used, the technique described by George W. Jacoby being followed (*N. Y. Med. Jour.*, Dec. 28, 1895–Jan. 4, 1896). As a rule, the fluid escaped readily, either in a jet or more or less quickly, drop by drop. In several cases the needle appeared to be clogged, gentle aspiration was practiced, the obstruction removed, the fluid escaping without further difficulty.

In one, lysol injections had been resorted to, the fluid following being of a gelatinous consistency; in the others, as soon as some flakes of fibrin were drawn off turbid fluid followed.

Lumbar puncture was performed in 49 cases. It was done early in 33 patients, late in 16, and repeatedly in 17 cases. In a case in which chronic hydrocephalus occurred (as a sequel), 17 punctures were made.

The fluid obtained by early lumbar puncture was usually quite turbid, frequently spurting out, as though under considerable pressure.

In several cases only a few drops of thick pus, chiefly polynuclear leucocytes containing numerous diplococci, were obtained. In these the prognosis was unfavorable. In a few instances the slightly turbid fluid contained heavy flakes of fibrin. The latter were composed of polynuclear leucocytes closely packed in a fibrinous material. At times they clogged the needle, but were readily withdrawn by gentle aspirations, after which the fluid would flow again.

In *four cases* the fluid was quite clear, containing but few leucocytes.

In the first, punctured on first day, 5 drams were removed; diplococcus found.

In the second, punctured on third day, 5 drams were removed; diplococcus not found.

In the third, punctured on seventh day, 8 drams were removed; diplococcus found.

In the fourth, punctured on tenth day, 10 drams were removed; diplococcus not found.

The intracellular diplococcus was found in the polynuclear cells in the fluid obtained by lumbar puncture in 44 cases. In the remaining 7 punctured we failed to get fluid in 1 case on account of the struggles of the patient; in 5 the fluid was clear or only slightly turbid (four of the specimens were obtained late in the course of the disease); in the seventh (an early case), a few drops of fluid, containing some leucocytes, were withdrawn. In a young girl of fifteen years, admitted within twelve hours from the beginning of the trouble, the diplococcus was found in the cerebro-spinal fluid. In Willie G., six years old, the micro-organism was found by culture as late as the fifty-eighth day.

"Within the vestibule of a new therapeutic achievement," too much was expected from lumbar puncture. The results, in consequence, have been disappointing in many respects.

It is readily conceded that a method which enables us to remove fluid which compresses important structures, as the brain and cord, is of great value in relieving certain symptoms. More than this we cannot claim.

Resorted to early, the progress of the disease has not been aborted or shortened, recrudescences or relapses have not been prevented. Notwithstanding repeated tappings, chronic hydrocephalus has supervened.

It is true, in the latter class, plastic exudates may have occluded the communication between the ventricles and the subarachnoid space.

If the problem were a simple one, a question of the removal of fluid only (as in serous meningitis), no doubt there would be less scepticism as to results.

In epidemics of cerebro-spinal meningitis we find a plastic exudation, a fibrino-purulent product. The fluid may be clear, turbid, straw-colored, purulent, with fibrin flakes or even pure pus.

Furthermore, the brain and cord are also involved. Under such conditions, with Osler we must say: " We may wonder rather that recovery follows in any well-developed case."

The results obtained appear below in tabular form.

Cases in which lumbar puncture was not made are presented in *Table A.*

In *Table B* are found the cases in which punctures were performed early, the amount taken away, etc.

Table C represents cases in which hypodermoclysis (for the purpose of diluting toxins in the blood, etc.) were made, in addition to the puncture.

Table D, the lumbar puncture was done late in the course.

Table E presents in tabular form the number of times punctures were made in same patient.

We have here presented our experiences, unfortunate though they are, simply as a contribution to the study of the varying features of this epidemic. We have not attempted to reduce our mortality by eliminating malignant or otherwise necessarily fatal cases. The work is a portrayal of the various phases of the disease, with its high death rate, in the lower " Eastern Section."

A careful consideration of the course of the plague and a study of the pathological lesions lead to the belief that in future preventive measures rather than remedial agents will overcome the dangers of the much dreaded epidemic cerebro-spinal meningitis.

At the time this article went to the printer there remained in the wards 18 cases. Of these 10 have since died, 5 have recovered, 3 were taken home unimproved. The total number of deaths was therefore 35, of recoveries 22.

The case of longest duration was in a girl of 8 years, who died on the 235th day of the disease.

TABLE A.

Nine Cases Without Puncture.

AGE.	RESULTS.	
3 years	Death on 2d day	*Post mortem puncture few drops of pus.
3 "	" 9th "	
3 "	" 12th "	Post mortem puncture. No fluid.
5 "	" 3d "	" " " "
6 "	Discharged 20th day..	Recovery complete.
12 "	" 68th " ..	Total deafness persisting.
16 "	Death 5th " ..	
16 "	Discharged 52d " ..	Recovery complete.
25 "	" 51st " ..	"

* In above cases for reason given in text, lumbar puncture was not resorted to.

TABLE B.

Early Lumbar Punctures—Thirty-five Cases.

1ST DAY.			2D DAY.			3D DAY.			4TH DAY.		
Age.	Amt. in fl. ʒ	Result.	Age.	Amt. in fl. ʒ	Result.	Age.	Amt. in fl. ʒ	Result.	Age.	Amt. in fl. ʒ	Result.
2 yrs..	5	Rec'y.	10 mos.	5	Death.	2 yrs..	5	Ward.	10 mos	Few drops	Ward.
3 " ..	4	Death.	1 yr..	4	"	3 " ..	8	Death.	10 "	13	Death.
7 " ..	4	Ward.	1 " ..	6	Ward.	3 " ..	5	"	3 yrs	Few drops	"
8 " ..	7	"	4 yr..	2	Rec'y..	3 " ..	5	Ward	3 "	Few drops	"
10 " ..	1.5	"	5 " ..	7	Ward.	4 "	13	Death.	3 " ..	0.5	"
15 " ..	3	Death.	5 " ..	4	Rec'y.	7 " ..	5	Rec'y.	6 "	8	Rec'y
16 " ..	10	Rec'y.	6 " ..	0.5	Death.	12 " ..	8	Death.	19 "	0	"
40 " ..	13	Death.	6 " ..	4	Rec'y.	15 " ..	0	"	24 " ..	8	Death
....	..	....	7 " ..	14	"	.....	..	......	.....	..	......
....	..	....	16 " ..	3	"	....	..	......	....	..	....
....	..	......	21 " ..	6	Death.	....	..	......	....	..	......
Av'ge..	5.93	....	....	53	....	....	6.13	....	....	4.33	....

TABLE C.

Early Lumbar Puncture with Hypodermoclysis—Eleven Cases.

SPINAL PUNCTURE.			HYPODERMOCLYSIS.		TERMINATION.
Age.	Day.	Amount in fl. ʒ	Day.	Amount in fl. oz.	
5 years	2d	4	3d	6	Recovery.
10 years	1st	1.5	1st	10	"
10 months	4th	13	4th	6	Death.
1 year	2d	4	3d	6	"
5 years	2d	7	2d	5	Ward.
3 years	3d	5	3d	6	"
4 years	2d	2	2d	10	Recovery.
7 years	1st	4	1st	10	Ward.
1 year	2d	6	2d	5	"
21 years	2d	6	2d	16	Death.
3 years	1st	4	1st	10	"

TABLE D.

Late Lumbar Punctures—Sixteen Cases.

AGE.	DAY.	AMOUNT IN FL. ʒ	TERMINATION.
8 months....	12th	2	Death.
4 years	15th	6	"
3 years.........................	8th	4	Recovery,
6 years......	17th	8	Death.
19 years......	19th	5	"
11 years.	6th	8	"
15 years........................	58th	6	Ward.
30 years.......	14th	12	Recovery.
5 years.........................	5th	4	Death.
1 year.....	7th	8	"
2 years.........................	10th	5	"
1 year...........................	10th	8	Recovery.
4 years.........................	9th	14	Death.
15 years........................	7th	1.5	Ward.
9 years............	11th	Few drops	"
2 years.........................	28th	5	"

TABLE E.

Repeated Lumbar Punctures—Seventeen Cases.

AGE.	DAY.	AMOUNT IN FL. ʒ.	TERMINATION.	PUNCTURES.
3 years	8th	4	Recovery	2
	11th	1		
7 years	17th	8	Ward	12
	28th	7		
	34th	10		
	52d	12		
	58th	8		
	63d	2		
	73d	7		
	74th	7		
	77th	3		
	80th	9		
	84th	5		
	91st	8		
15 years	3d	..	Ward	17
	5th	8		
	6th	9		
	7th	1		
	23d	12		
	25th	7		
	27th	7		
	28th	7		
	30th	..		
	34th	14		
	41st	2		
	47th	..		
	62d	4		
	64th	Few drops		
	66th	Few drops		
	66th	3		
19 years	19th	5	Death	3
	20th	8		
	26th	2		
8 years	1st	7	Ward	2
	22d	7		
3 months	4th	Few drops	Death	6
	6th	Few drops		
	8th	4		
	21st	4		
	28th	..		
	33d	..		
10 months	2d	5	Death	2
	4th	9		
2 years	4th	0.5	Death	2
	10th	1		

TABLE E—*Continued.*

Repeated Lumbar Punctures—Seventeen Cases.

AGE.	DAY.	AMOUNT IN FL. ʒ.	TERMINATION.	PUNCTURES.
16 years	1st	10	Recovery	2
	2d	11		
5 years	2d	4	Recovery	2
	4th	8		
4 years	3d	13	Death	5
	6th	5		
	9th	4		
	15th	7		
	19th	5		
15 years	4th	..	Ward	2
	7th	1.5		
9 years	11th	Few drops	Ward	4
	16th	7		
	18th	4		
	19th	5		
7 years	1st	4	Ward	2
	5th	4		
2 years	28th	5	Ward	2
	35th	5		
2 years	3d	5	Ward	2
	11th	9		

NAME, AGE, ADDRESS, ADMITTED.	ONSET.	Day of Disease.	CONDITION ON ADMISSION.	COMPLICATION.
1. Annie K., 12 yrs., 640 E. 5th st., Feb. 21, 1904...	Headache (occip.); restlessness; vomiting; very talkative; irrational 2d day	2d	Excitable; irrational at times; completely deaf; neck rigid; Kernig; T. 103, P. 126, R. 28........	Total deafness.....
2. Carmelia R., 8 mos., 127 Canal st., Mar. 8, 1904	Fretful; vomiting; green stools.....	3d	Stupor; pupils inactive; neck rigid; Kernig; T. 98, P. 120, R. 30........	Vomiting recurred and persisted late in course..........
3. Sadie L., 16 yrs., 116 Suffolk st., Mar. 11, 1904..	Vomiting; pains in abdomen; becoming localized to right of umbilicus; headache (frontal).........	4th	Mental condition clear; cheeks flushed; conjunctiva injected; lies on side with thighs flexed; refers pain to right of umbilicus, where she is quite tender...........	Herpes 5 d.; pneumonia, signs apparent 6 d.; no cough or expectoration; (right upper lobe)....
4. Sam'l K., 16 yrs., 106 Orchard st., Mar. 12, 1904..	Headache; restlessness.........	2d	Unconscious; pupils inactive; neck rigid; Kernig; unable to swallow; T. 100/4, P. 88, R. 28...........	Nephritis; 10 per cent. albumen..
5. William L., 4 yrs., 654 Water st., Mar. 14, 1904..	Restlessness; vomiting...........	2d	Unconscious; pupils inactive; Kernig; head retracted; T. 100, P. 108, R. 32.	Herpes 2 d.; conjunctivitis, rhinitis (smears and cultures negative), constipation... ....
6. Grace L., 3 yrs., 367 Cherry st., Mar. 26, 1904..	(Two other children in family have same disease) headache; restlessness; vomiting...	2d	Stupid, irritable, pupils inactive, neck rigid; Kernig; vomiting; T. 103/4, P. 148, R. 48	Herpes 3d d.; conjunctivitis, rhinitis, bronchitis, constipation............
7. William G., 6 yrs., 73 Willet st., Mar. 15, 1904...........	Headache; vomiting; restlessness.	3d	Stupid, irritable, neck rigid, pupils inactive; Kernig; herpes; T. 104/6, P. 130, R. 48.....	Herpes 3d d.; Bronchitis late in course, hydrocephalous, constipation late in course, diarrhœa.
8. Joseph R., 19 yrs, 135 Norfolk st., Mar. 21, 1904........	Headache; restlessness; hallucinations alternating with stupor, this condition lasting two weeks; convulsions and delerium on 13th day.............	15th	Unconscious; irritable; pupils dilated, inactive; neck rigid; Kernig; T. 101, P. 80, R. 30	Vomiting occurred during course; constipation ...

SPINAL PUNCTURES AND HYPODERMOCLISIS.	REMARKS.	RESULT.
....	Temp. irreg., continuous till 7th day. Intermittent until 34th day, then normal except for slight exacerbation, 44th to 50th day. Temp. range, 98 to 104. Pulse, 80 to 128....	Recovery complete except for deafness. Discharged 68th day.
S. P., 12th d., 2 dr., turbid, Dip. —................	Low temp., and stupor throughout. Marked emaciation. Post Mortem—brain soft, purulent exudate over entire surface, most marked over vertex (here collections of fibrin); considerable turbid fluid in ventricles and canal.......	Death: 45th day. Marasmus, Hydrocephalus.
....	A few hours after admission, operated upon for appendicitis (normal appendix). On next day no abdominal pain, but severe frontal headache. Neck rigid, slight retraction of head. Kernig. (Wound healed primarily.) Temp. for first two weeks, high with remissions, 100 to 105. Had several chills at irregular intervals. During second week, temp. intermittent, normal after 32d day. All rigidity gone by 35th day. Mental condition clear throughout.....	Recovery complete. Discharged 52d day.
....	Temp., 100 to 102/8. Pulse, 88 to 120. Unconscious throughout.............	Death; 5th day.
S. P., 15th d., 6 dr., slightly turbid, Dip. —..........	Improvement after first few days, then relapse with high remittent temp., 101 to 105. Pulse, 96 to 140. Stupid and irritable throughout; great emaciation..	Death; 42d day. Marasmus.
S. P., 8th d., 4 dr., Dip. +; S. P., 11th d., 1 dr.......	Restlessness during first few days gave place to stupor and irritability. Temp. high with remissions and intermissions, 96 to 105 till 19th day. Pulse 92 to 150. All rigidity gone by 30th day..........	Recovery; discharged 42d day. Considerable emaciation, somewhat stupid.
S. P., 17th d., 8 dr., slightly turbid, Dip.+; S. P., 28th d., 7 dr., slightly turbid; S. P., 34th d., 10 dr., slightly turbid; S. P., 52d d., 12 dr., slightly turbid, Dip.+; S. P., 58th d., 8 dr., slightly turbid; S. P., 63d d., 2 dr., slightly turbid; Hypo. of saline., 71st d., 8 oz.; Hypo. of saline., 73d d., 7 oz.; S. P., 73d d., 7 dr., slightly turbid; S. P., 74th d., 7 dr., slightly turbid; S. P., 77th d., 3 dr., slightly turbid..................	Temp. high with intermissions until 11th day, then normal until 20th day. During this period steady improvement. Temp. then became quite irregular until 35th day. Patient steadily failing, stupid, at times restless and delirious; after 35th day slight occasional elevations of Temp. Emaciation marked, though large quantities of nourishment taken. Mental condition late in course varied greatly, at times fairly bright, few moments later could not be aroused. No immediate results could be attributed to the spinal punctures. Pupils widely dilated, inactive, or contracting and dilating under light (Opthalmoscopic exam. neg.). Pulse range 80 to 134...........................	Still in ward 88th day.
S. P., 19th d., — 5 dr., clear, Dip. — T.B.; S.P., 2toh d., 8 dr., clear, Dip.—; S. P., 26th d., 2 dr., clear, Dip. —.................	Temp. normal throughout, *i. e.*, during time in hospital, save for occasional slight elevations. Pulse 80 to 120. Stupor alternating with restlessness and delirium. Emaciation marked....	Death; 32d day. Exhaustion.

NAME, AGE, ADDRESS, ADMITTED.	ONSET.	Day of Disease.	CONDITION ON ADMISSION.	COMPLICATION.
9. Max P., 15 yrs., 18 Forsyth st., Mar. 26, 1904..	Headache; vomiting; restlessness	3d	Very restless; semiconscious; pupils small, inactive; neck rigid; Kernig; herpes; T. 103, P. 126, R. 24.	Herpes 3d d.; strabismus, bronchitis, conjunctivitis, Dip. + ...
10. Rosie S., 11 yrs., 100 Forsyth st., Mar. 27, 1904..	Headache; vomiting............	3d	Delirious; restless; hyperaesthesia marked; neck rigid; Kernig; T. 101, P. 118, R. 24.	None............
11. Moses G., 12 yrs., 64 Delancey st., Mar. 28, 1904..	Headache; pain in abdomen; vomiting; convulsions.	1st	Delirious; pupils dilated and inactive; neck rigid; Kernig; T. 104, P. 72, R. 20......	None..............
12. Loretta D., 6 yrs., 11 Hamilton st., April 2, 1904.....	Vomiting; headache; convulsions. (Two other cases in same house)...	2d	Semi-conscious; restless; irritable; vomiting; head retracted; Kernig; T. 101, P. 120, R. 26............	Conjunctivitis, rhinitis, constipation. ....
13. Bertha S., 3 yrs., 36 Forsyth st., April 5, 1904...	Vomiting; headache; restlessness	2d	Coma (moribund); rigid back, neck and extremities; T. 105, P. 150, R. 36; petechial.....	Conjunctivitis, rhinitis ....
14. Dan D., 25 yrs., 65 Gouverneur, April 6, 1904...	Chill; headache (occip.); general body pains; vomiting 3d day.....	4th	Apathetic; pupils normal; facial twitchings; no rigidity; T. 102, P. 96, of high tension; ,slight oedema of legs...	Nephritis - parotitius (bilateral) 17th d. to 25th d.
15. Fred S., 3 yrs., 11 Chambers st., Apr. 9, 1904	Headache; vomiting; delirium...	3d	Coma; eyes sunken; pupils inactive; head retracted; lordoris: Kernig; T. 102, P. 96, R. 28	Constipation ....
16. Guatano C., 3 yrs., 139 Cherry st., Apr. 9, 1904.	(Sister brought to hospital with same disease). Vomiting; "Spots" 2d day; convulsions 4th day; herpes 4th day.............	9th	Apathy; cyanosis; dysponea; eyes sunken; pupils sluggish; neck rigid; no Kernig; T. 101, P. 123, R. 46; Pneumonia (broncho); mottled extremities.......	Broncho - Pneumonia (double) on admission ..

Spinal Punctures and Hypodermoclisis.	Remarks.	Result.
S. P., 3d d., no fluid; S. P., 5th d., 8 dr., turbid, Dip. +; S. P., 6th d., 9 dr., Lysol ½ per cent., 3 dr. injected; S. P., 7th d., 1 dr., turbid, Lysol ½ per cent., 1 dr. injected; S. P., 23d d., 12 dr., slightly turbid; S. P., 25th d., 7 dr., slightly turbid, Dip. +; S. P., 27th d., 7 dr., slightly turbid, Lysol 3 per cent., 7 dr. injected and withdrawn; S. P., 28th d., 7 dr., slightly turbid; S. P., 29th d., 11 dr.; S. P., 30th d., no fluid; S. P., 34th d., 14 dr.; S. P., 41st d., 2 dr., clear; S. P., 47th d., no fluid; S. P., 62d d., 4 dr.; S. P., 64th d., 20 minims; S. P., 66th d., 2 minims; S. P,, 66th d., 3 dr.........	Temp. high with remissions and intermissions until 35th day. Restlessness of first few days giving place to stupor, with dilated pupils; marked retraction of head. After 35th day steady improvement, mental condition clear, rigidity disappeared. Sight and hearing good, allowed to sit up. Pupils remained somewhat dilated and sluggish. After 55th day complained of headache; vomiting after eating. Mental condition varied greatly, at times bright, and a few hours later stupor. 65th day, hiccoughs, controlled by morphine. No immediate results of the spinal puncture could be noted..	Death; 56th day. After respiration ceased, pulse continued good for two or three minutes. Lumbar puncture done, 3 dr. removed and artificial resp. resorted to. Pulse continued for about ten minutes.
S. P. 6th d., 8 dr., very turbid, Dip +..........	Semi-conscious and reatless throughout. Temp., 98 to 102. Pulse, 104 to 140....	Death; 7th day.
None....................	Unconscious throughout; convulsions. Temp., 96 to 104. Pulse, 60 to 150.....	Death; 3d day.
None....................	Steady improvement from time of admission. Vomiting stopped third day. Fourth day became very talkative. Temp. normal after 4th day. No rigidity after 12th day.....	Recovery complete. Discharged 20th day.
S. P., post mortem, few drops of pus, Dip. +....	....	Death; immediately after admission.
None....................	Intense pain in back of head and neck most marked symptom. Rigidity of head and Kernig not noted until several days after admission. Disappeared by 22d day. Temp., irreg., 99 to 103, until 18th day, then normal. Pulse, 52 to 108..................................	Recovery complete. Discharged 51st day.
S. P., 4th d., few drops of pus, Dip. +............	Unconscious throughout. Temp, 101 to 105. Pulse, 96 to 150.................	Death; 5th day.
....	Apathetic and unconscious throughout. Temp., 101 to 103. Pulse, 122 to 150. Resp. 40 to 50.......................	Death; Pneumonia; 12th day.

NAME, AGE, ADDRESS, ADMITTED.	ONSET.	Day of Disease.	CONDITION ON ADMISSION.	COMPLICATION.
17. Jane C., 15 yrs., 139 Cherry st., Apr. 9, 1904....	(Sister brought to hospital at same time, with same disease). Vomiting; headache..	10th	Mental condition clear; pupils normal; head retracted; lordoris; Kernig; T. 102/6, P. 100, R. 44......	Bronchitis.......
18. Jake E., 30 yrs., 217 Bowery, Apr. 9, 1904....	Headache; vomiting.............	4th	Neck rigid; tender swelling; beneath left sterno-mastoid; pharyngitis; pupils normal; Kernig; T. 104/6, P. 76, R. 24......	Conjunctivitis; Dip. —; Pharyngitis, Adenitis (cervical), constipation....
19. Fannie H., 8 yrs., 42 Chaystie st., Apr. 13, 1904	Vomiting; restless; delirious; retraction of head............	12 hrs. after onset.	Delirious; very restless; head retracted; pupils dilated and sluggish; marked photophobia; Kernig; lordoris; T. 98, P. 96, R. 24............	Herpes 3d d.; conjunctivitis, 1st d. dip. +; rhinitis 1st d.; bronchitis and haemorrhages around wrist joints and dorsum of hands 3d d., resolving in few days; pain and tenderness around left shoulder 14th d.; contracture persists; partial atrophy of optic discs noted, 27th d........
20. Frank L., 16 yrs., Salvation Army Hotel; Apr. 13, 1904..	Severe headache; malaise and pains in back....	2d	After admission had chill and became comatose; neck rigid; pupils dilated and sluggish; T. 102, P. 108, R. 20; Kernig; few petechiae.	Strabismus 3d d.; persisted; conjunctivitis, Dip.+.........
21. Mamie M., 5 yrs., 17 Forsyth st., Apr. 15, 1904..	Vomiting; convulsions.	3d	Coma (moribund); Kernig, no other rigidity; T. 98, P. 120, R. 32....	Conjunctivitis, Dip.+.........
22. David L., 3 yrs., 191 Allen st., Apr. 15, 1904..	Vomiting; convulsions (one other case in house)...	3d	Unconscious; restless; eyes sunken; pupils inactive; head retracted; Kernig; T. 99, P. 102, R. 30; few petechiae........	Conjunctivitis, Dip.,+ rhinitis, bronchitis, strabismus ....
23. Rebecca S., 7 yrs., 22 Norfolk st., Apr. 19, 1904...	Headache; vomiting; convulsions	2d	Unconscious; restlessness (shock); pupils small; inactive; neck slightly rigid; Kernig; pulse and resp. irregular; T. 103/4, P. 122, R. 28; profuse petechiae	Paralysis of right side of face from 3d to 6th day..

Spinal Punctures and Hypodermoclisis.	Remarks.	Result.
S. P., 58th d., 6 dr., slightly turbid, Dip. +..........	Mental condition has remained clear. General hyperaesthesia has been marked and persistent. Temp. high and irregular, 100 to 104, till 30th day, continues somewhat lower until present. Pupils somewhat dilated, sluggish. Emaciation..........................	Still in hospital 60th day of illness.
S.P., 14th d., 12 dr., slightly turbid, Dip. +..........	Mental condition clear throughout. Temp. remit. high, 100 to 105, till 15th day. Pulse slow, 60 to 100. Adenitis resolved by 15th day. After 25th day no symptoms save indefinite body pains..	Recovery complete. Discharged 51st day.
S. P., 1st d. on admis., 7 dr., turbid, Dip.+, 22d d. 7 dr., slg. turbid, Dip,+.	Montal condition cleared in a few days. Temp. irreg , 98 to 104, persists to present time. Pulse, 92 to 120. Mental condition remains clear, pupils quite variable. Considerable emaciation. Vomiting recurred after 35th day. Has severe headaches.....................	In ward 55th day, failing steadily.
S. P., 2d d., on admis , 8 dr., turbid, Dip.+......	Eight hours after puncture semi-conscious and restless. On 3rd day mental condition clear, pupils active. head retracted. Steady improvement with the temp, irreg., remittent and intermittent, 101 to 102 for 17 days. No rigidity after 17th day. Opthalmoscopic examination. O. D. S. Discs indistinct. (Specific ?)..........	Recovery complete, save for strabismus, discharged 41st day.
S. P., post mortem, no fluid.	....	Death 3d day 2 hours after admission.
....	Unconscious throughout. For 36 hours before death, constant twitchings of right half of body. Temp., 98 to 102. Pulse, 92 to 120......................	Death ; 9th day.
S. P., 2d d., on admis. 4 dr., turbid, Dip.+.......	Steady and rapid improvement. On third day conscious and quiet. No temp. after 12th day. No rigidity after 15th day. Temp., 97 to 103/4. Pulse, 92 to 122	Recovery complete. Discharged 20th day.

Name, Age, Address, Admitted.	Onset.	Day of Disease.	Condition on Admission,	Complication.
24. Solomon F., 6 yrs., 92 Willet st., Apr. 21, 1904..........	Headache; vomiting; convulsions	2d	Unconscious; restless; eyes sunken; pupils dilated, inactive; strabismus; neck rigid; Kernig; T. 102, P. 88, R. 26; numerous petechiae and mottled extremities........	Rhinitis, conjunctivitis, constipation..........
25. Lewis F., 5 yrs., 196 Stanton st., Apr. 22, 1904...	Vomiting; headache; restlessness............	5 h	Unconscious; restless; eyes sunken; pupils dilated, inactive; strabismus; neck rigid; Kernig; T. 102, P. 80; R. 28........	....
26. Dolly K., 6 yrs., 92 Attorney st., Apr. 22, 1904..	Pain in knee; headache; vomiting..	2d	Coma; marked pallor (shock); pupils inactive; Kernig; no rigidity of neck; T. 97, P. 92 (feeble); R. 24; mottled extremities..............	Conjunctivitis, rhinitis; herpes 3rd day; constipation.......
27. Dominic G., 19 yrs., 78 Mott st., Apr. 22, 1904..	Headache; vomiting,............	3d	Mental condition clear; face flushed, slight conjunctivitis; pupils normal; herpes; neck rigid; Kernig; T. 100, P. 88, R. 20.........	Conjunctivitis; Dip. + herpes 3rd day........
28. Celia L., 3 yrs., 83 Norfolk st., Apr. 24, 1904 ..	Vomiting; headache; convulsions...........	3d	Unconscious; restless; pupils inactive; head retracted; Kernig; T. 102, P. 100, R. 26; few petechiae	Conjunctivitis; Dip. + constipation..........
29. Sarah D., 2 yrs., 277½ Madison st., Ap. 24, 1904	Vomiting; convulsions............	3d	Conscious; irritable; pupils sluggish; neck rigid; Kernig; T. 101/8, P. 112, R. 28.....	Conjunctivitis, rhinitis........
30. Esther M., 2 yrs., 94 Rivington st., April 26, 1904..	Restlessness; headache; vomiting; loss of consciousness...........	1st 8 hrs.	Unconscious; restless; delirious; marked photophobia; pupils dilated and sluggish; no rigidity; T. 102, P. 106, R. 28.	Conjunctivitis; Dip + vaginitis, (staphylococcus.)..........
31. Alex G., 3 yrs., 116 Suffolk st., April 28, 1904..	Headache; vomiting; delirium....	3d	Unconscious; restless; eyes sunken; pupils inactive; head retracted; lordosis; Kernig; slight cyanosis; T. 101, P. 99, R. 28; skin mottled.	Conjunctivitis....
32. Jennie W., 7 yrs., 402 Madison st., April 29, 1904..	Headache; vomiting; convulsions.	3d	Conscious; restless; pupils normal; slight rigidity of neck; slight Kernig; T. 104/4, P. 140, R. 28.	Rhinitis, Dip—..

SPINAL PUNCTURES AND HYPODERMOCLISIS.	REMARKS.	RESULT.
S. P., 2d d., 5 dr., very turbid, Dip. +..........	Left hemiplegia on 3d day, persisting. Unconscious throughout. Temp. irreg., 27 to 105. Pulse 88 to 160........	Death; 5th day.
S. P., 5th d., 4 dr., very turbid, Dip.+..........	Ante-mortem temp. 107..................	Death; 4 hours after admission.
S. P., 2d d., on admis., 4 dr., turbid, Dip. +......	Conscious and restless a few hours after puncture with head retracted and neck rigid. Temp. 100 to 103 for 5 days, then normal. Rapid and steady improvement from time of admission. No rigidity after 7th day................	Recovery complete. Discharged 20th day.
S. P., 4th d., no fluid, patient resisted and puncture made with difficulty..............	Steady and rapid improvement. Temp, irreg. to 104, normal after 10th day. No symptoms after 12th day. Pulse, 64 to 100..................................	Recovery complete. Discharged 33d day.
S. P., 3d d., 5 dr., very turbid, Dip. +..........	Unconscious throughout. Temp., 101 to 204. Pulse, 90 to 118..................	Death; 5th day
S. P., 3d d., 5 dr., very turbid, Dip. +; 11th d., 9 dr., slg. turbid, Dip. +.................	Stupid and irritable throughout. Temp. irreg., 98 to 103. Pulse, 94 to 124. Pupils dilated and sluggish; considerable emaciation. After 36th day mental condition brightening; pupils respond better; gaining weight; slight elevations of temp....................	In ward 43d day of disease; improving.
S. P. 1st d., 5 dr., clear with few leucocytes Dip.+. :	Conscious and irritable on 3d day. No rigidity of neck, no Kernig at any time. Photophobia until 5th day. Low Temp., 100 to 101 till 6th day.	Recovery complete. Discharged 20th day.
S. P. 3d d., 8 dr., turbid, Dip.+.................	Unconscious throughout. Ante-mortem temp. 106..........................	Death 4th day.
S. P. 3d d. 5 dr., clear Dip.— few leucocytes..........	Steady improvement, mental condition clear throughout. Rigidity only slight, none after 11th day. Temp. 98 to 105, normal after 10th day. Pulse, 82 to 140.	Recovery complete. Discharged 16th day.

Name, Age, Address, Admitted.	Onset.	Day of Disease.	Condition on Admission.	Complication.
33. Ida G., 3 yrs., 35 Market st , Apr. 29, 1904........	Headache ; vomiting ; T. 106 ; convulsions 3d day..	4th	Semi-conscious ; pupils sluggish ; neck rigid ; strabismus ; Kernig ; T. 108/8, P. 132, R. 32...........	Conjunctivitis, Rhinitis, Bronchitis, Strabismus ....
34. Wm. B., 40 yrs., 15 Bowery, Apr. 30, 1904...	Found unconscious in bed of lodging house; no definite history obtainable.........	1st	Unconscious ; pupils small, inactive ; head slightly retracted ; Kernig ; T. 102, P. 130, R. 36 ; profuse petechia.....	....
35. Edward D., 10 mos., 395 Water st , May 1, 1904	Restlessness; vomiting ; stupor....	2d	Stupor ; pupils sluggish ; neck rigid ; fantanelle tense ; no Kernig ; T. 103 ; P. 140, feeble, R. 50, irregular ; cyanosis, petechia........	Conjunctivitis, Dip. +........
36. Mary K., 6 yrs., 11 Hamilton st., May 1, 1904....	Headache (frontal); vomiting 2d d.; convulsions 3d d.; (two other cases in same house)...	4th	Stupor; pupils sluggish; herpes profuse; head retracted; Kernig; T. 101/4, P. 120, R. 26...........	Rhinitis; bronchitis.........
37. Lena D., 2 yrs , 120 Ludlow st., May 2, 1904.....	Vomiting; headache; convulsions 3d d............	4th	Conscious; irritable; pupils sluggish; head retracted; Kernig; T. 103/4, P. 118, R. 28...........	Conjunctivitis. Haemorrhage around wrist on adm., slowly resolving haematoma...........
38. Maggie P., 16 yrs., 128 Cherry st., May 3, 1904.	Headache; unconscious; vomiting; delirious in 7 hrs.	1st, 14 hrs.	Coma; pupils dilated and inactive; vomiting; neck rigid; Kernig; leucocytes 30,000; T. 103, P. 132, fair; R. 24, irreg.; mottled extremities.......	Conjunctivitis, herpes 3d d...
39. Morris S., 5 yrs., 76 Eldridge st., Apr. 4, 1904....	Chill ; headache ; vomiting ; convulsions 2d day..	2d	Unconcious ; restless ; face flushed, cyanosed ; eyes sunken, pupils dilated, inactive ; head retracted ; Kernig ; T. 102, P. 120, feeble, R. 32...............	Conjunctivitis ; Dip. +, stomatitis ....

SPINAL PUNCTURES AND HYPODERMOCLISIS.	REMARKS.	RESULT.
S. P. 4th d., few drops pus, Dip. +; S. P. 6th d., few drops pus, Dip. +, lysol, ½ per cent., 6 dr.; injected, 3 dr.; 7th d., 6 oz. hypo. of saline sol.; S. P. 8th d., 4 dr. turbid with fibrine flakes; 13th d., 6 oz. hypo. of saline sol.; 15th d., 6 oz. hypo. of saline sol.; 17th d., 6 oz. hypo. of saline sol.; S. P. 21st d., 4 dr. turbid, Dip. —; 27th d., 6 oz. hypo. of saline sol.; S. P. 28th d., no fluid; S. P. 33d d., no fluid.............	The spinal fluid contained heavy lumps of fibrin, which would clog needle, stopping flow; if these were withdrawn by aspirator, fluid would at times flow freely. After injection of lysol on 6th day, child became much worse; pulse feeble, abdomen distended; lysol was withdrawn through larger needle after about 4 hours; fluid quite gelatinous, making aspiration necessary. Patient failed steadily. On two occasions, oedema of lungs, eyes glazed, pulse very feeble; sub-cutaneous injection of saline given, (6 oz.); patient rallied. Temp. continuous during first 2 weeks, becoming remittent and irreg., 98 to 100. Pulse, 108 to 170. Emaciation marked. Became totally blind, deaf and unable to swallow................	Death; 39th day.
S. P. 1st d., on admis. 13 dr. turbid, Dip.+..........	Grew worse steadily; had three convulsions during 9 hours in hospital.......	Death; 1st day.
S. P. 2d d., 5 dr. turbid, Dip. +; 4th d., 9 dr. turbid, Dip. +..........	Somewhat brighter for short time on 3d day (Kernig present), but soon failed. Fontanelle tense and bulging before 2d puncture, afterwards soft, otherwise no change noted. Temp. 102 to 106, Pulse, 140 to 160......................	Death; 5th day.
S. P., 4th d., 8 dr., slg. turbid, with heavy flakes of fibrin, Dip +........	Temp. low; normal after 10th day. Steady improvement. No rigidity after 11th day............................	Recovery complete. Discharged 18th day.
S. P., 4th d., ½ dr., slightly turbid, Dip. +; 10th d., 1 dr., slightly turbid, Dip. +................	Mental condition remained clear. Temp, high, with remissions, 100 to 105. Pulse, 108 to 140. Rigidity gone; pupils normal by 24th day. Shows some emaciation. About 33d day began to fail rapidly; convulsions, more violent on left side; became rigid, unconscious, remaining so..................	Death; 36th day.
S. P., 1st d., on admis., 10 dr., turbid, Dip. +; 2d d., 11 dr., turbid, Dip. +	Ten hours after first puncture, semi-conscious and restless; pupils better. On 2d day very restless; pupils again inactive. On 3d day mental condition clear, patient quiet, pupils reacting fairly well. Leucocytes, 13,500. Vomiting persisted till 3d day. Temp. steadily declined to normal on 5th. No symptoms after 12th day. Opthalmoscopic examination 12th day. O. D. S. some oedema of discs.........	Recovery complete. Discharged 28th day.
S. P., 2d d., 4 dr., turbid, Dip. +; 3d d., 6 oz. hypo. of saline; S. P., 4th d., 8 dr., very turbid, Dip. +; 4th d., 9 oz. hypo. of saline; 5th d., 6 oz. hypo. of saline..................	On 2d day, more quiet and conscious, but condition very bad until 5th day; pupils inactive, rapid emaciation with feeble pulse, oedema of lungs. After 5th day steady improvement, Temp. steadily declined to normal on 11th day and remained so. No symptoms after 17th day. Pulse, 96 to 124.................	Recovery complete. Discharged 18th day.

Name, Age, Address, Admitted.	Onset.	Day of Disease.	Condition on Admission.	Complication.
40. Henry G., 4 yrs., 13 Bayard st., May 8, 1904...	Vomiting; restless ness............	3d	Semi-conscious; restless; irritable; pupils inactive; head retracted; Kernig; T. 99, P. 88, R. 24, leucocytes, 25,000.....	Conjunctivitis; Dip. +, rhinitis, bronchitis......
41. Kate E., 15 yrs., 165 Eldridge st., May 8, 1904....	Found in bed by parents, semi-conscious; vomiting; had been well two hours previous........	1st	Unconcious; marked pallor; pupils dilated, inactive; neck slightly rigid: slight Kernig; T. 100/2, P. 108, R. 16. Pulse full and strong; respiration irregular.............	None............
42. Peter D., 24 yrs., 667 Water st., May 9, 1904....	Vomiting; pains in back; previous alcoholic debauch; was heavy drinker...	4th	Semi-conscious; mild delirium; pupils normal; tongue dry and brown; neck rigid; Kernig; T. 103, P. 120, R. 26.	Bronchitis on admis; conjunctivitis; Dip.—...
43. Ida S., 10 yrs.., 410 Water st., May 11, 1904..	Nausea; no vomiting, unconscious in 7 hours.......	1st. 8th hr.	Unconscious; marked pallor (shock); slight rigidity of neck; Kernig; pupils small and inactive; T. 103, P. 130, feeble, R. 34; numerous petechiae...............	Conjunctivitis 2 d.; herpes 3 d..
44. Samuel H., 10 mos., 45 Eldridge st., May 5, 1904.........	Vomiting; fretful; stupor.........	4th	Coma; pallor; pupils dilated and inactive: nystagmus; fontanelle tense bulging; trismus; unable to swallow; stertorous irregular breathing........	Conjunctivitis....
45. Lillie R., 1 yr., 69 Henry st., May 12, 1904..	Vomiting; convulsions, 1st d......	2d, 28 hrs.	Semi conscious; restless; pupils inactive; slight rigidity of neck; Kernig; fontanelle normal; T. 103, P. 160, feeble, R. 48, irregular; numerous petechiae; mottled extremities.......	Conjunctivitis; Bronchitis.....

SPINAL PUNCTURES AND HYPODERMOCLISIS.	REMARKS.	RESULT.
S. P., 3d d., 13 dr., very turbid, Dip. + ; S. P.. 6th d., 5 dr., very turbid, Dip. + ; S. P., 9th d., 4 dr., very turbid, Dip. + ; 15th d., 10 oz. hypo. of saline ; S. P., 17th d., 7 dr., very turbid ; 17th d., 8 oz. hypo. of saline ; 18th d., 10 oz. hypo. of saline ; S. P., 19th d., 5 dr., turbid, Dip. +.............	Mental condition cleared in few days, but otherwise grew steadily worse. Temp. high, irreg., with occasional chills, rigidity and emaciation increasing. On 15th day had convulsion of left side, lasting 3 hours, oedema of lungs ; 7 drams of spinal fluid removed, and hypo. of saline, 10 oz., during convulsions. Stimulation with caffeine ; boy rallied. Oedema recurred several times, but cleared up under caffeine sodium benzoate. Remained conscious until 18th day. Pupils dilated and inactive. Great emaciation. Temp., 98 to 105. Pulse, 88 to 170......................	Death ; 23d day.
S. P., 1st d. on admis. 3 dr., turbid, Dip. +..........	Became very restless shortly after puncture, but still unconscious. About 8 hours after admission breathing became slow and irregular. Pulse remaining good for several moments after respiration had ceased................	Death ; 15 hours after onset.
S. P., 4th d., 8 dr., clear, Dip+ ; 8th d., 1 pint hypo. of saline ; 10th d., 1 pint hypo. of saline ; 11th d., 1 pt. hypo. of saline..................	Steady failure, semi-conscious and delirious throughout. Temp. 100 to 103. Pulse 100 to 140. Leucocytes 6th day 13,200	Death ; toxaemia and delirium tremens, 12th day.
S. P., 1st d.. on admis., 1½ dr., turbid, Dip.+ ; 1st d., 10 oz. hypo. of saline..	A few hours after admission, became restless and irritable, pulse better on 2d day; conscious and very restless, rigidity more marked. On 3d day quiet, mental condition clear, pupils normal. Temp. course was irreg., low, ranging from 99 to 103/4. Pulse 90 to 148. The temp. remained practically normal after 6th day. No rigidity after the 10th day. Leucocytes on 3d day, 18,200, on 6th day, 11,500.............	Still in the ward on the 18th day, no symptoms........
S. P., 4th d., 8 dr., turbid, Dip.+ ; 4th d., 6 oz. hypo. of saline...........	Fontanelle soft, and breathing better after puncture. Remained in coma. Temp. 103 to 106. Pulse 180 to 190...	Death ; 5th day ;
S. P.. 2d d., admis., 4 dr., turbid, Dip. + ; 3 d., 6 oz. hypo. of saline ; 5 d., 8 oz. hypo. of saline....	Steady failure, remained unconscious. Vomiting persisted. On 6th day unable to swallow. Temp., 101 to 104. Pulse, 120 to 180. Rapid emaciation..	Death ; 6th day.

NAME, AGE, ADDRESS, ADMITTED.	ONSET.	Day of Disease.	CONDITION ON ADMISSION.	COMPLICATION,
46. Kate S., 1 yr., May 12, 1904..	Vomiting; convulsions; unconscious 3d d.........	7th	Coma; fontanelle slightly bulging; pupils dilated, inactive; nystagmus; no rigidity of neck; no Kernig; T. 98, P. 124, R. irreg., sighing.	Conjunctivitis; Rhinitis........
47. Fannie C., 2 yrs., 192 Division st., May 13, 1904...	Pains in leg., retraction of head, no appetite; vomiting 8th d.......	10th	Semi-conscious; irritable; pupils dilated: inactive; slight rigidity of neck; Kernig; T. 102, P. 128, R. 22, sighing and irregular.........	None............
48. Lizzie K., 11 mos., 37 Monroe st., May 13, 1904	(Brother in hospital with same disease); vomiting; diarrhoea; stupor	3d	Stupid; irritable; neck rigid; Kernig; fontanelle normal; pupils normal; T. 104/4, P. 140, R. 44.....	Conjunctivitis; Dip.+; rhinitis Dip. +; diarrhoea..........
49. Louis T., 1 yr., 61 Jefferson st., May 13, 1904..	Vomiting; restlessness; cough; "Spots," fading in few days	10th	Restless; irritable; head strongly retracted; no Kernig; fontanelle normal; pupils active; T. 101, P. 120, R. 28........	Rhinitis, Dip. —; conjunctivitis, Dip.—; bronchitis. ....
50. Joseph H., 4 yrs., 37 Monroe st., May 14, 1904 ..	(Sister in hospital with same disease); headache; vomiting; semi-conscious 3d day, remaining so; convulsions 5th day.............	9th	Unconscious; blind; deaf; pupils dilated and inactive; slight rigidity of neck; Kernig; T. 98, P. 102, R. 28; irreg.; sighing	None ....
51. Margaret R., 5 yrs., 202 Madison st., May 14, 1904.	(Sister in hospital with same disease.) Vomiting; headache; retraction of head; stupor	2d	Semi-conscious; restless; head strongly retracted; pupils inactive; marked photophobia; Kernig; T. 100, P. 98, R. 28; fem. petechiae.. ..	Conjunctivitis, bronchitis, herpes, 4th d......
52. Fannie M., 15 yrs., 278 E. 7th st., May 15, 1904..........	Headache; retraction of head; restlessness; delirium 3d day..........	4th	Conscious; restless; irritable; pupils normal; head strongly retracted; Kernig; T. 102/6, P. 120, R. 30............	Herpes 4th d.; conjunctivitis...
53. Catherine R., 3 yrs., 202 Madison st., May 15, 1904.	(Sister in hospital with same disease.) Vomiting; stupor...........	3d	Unconscious; irritable; head retracted; Kernig; pupils dilated, inactive; T. 99, P. 128, R. 40; fem. petechiae; mottled extremities..	Conjunctivitis; herpes, 6th d...

SPINAL PUNCTURES AND HYPODERMOCLISIS.	REMARKS.	RESULT.
S. P., 7th d., 8 dr., clear, Dip. + T. B............	Remained in coma. Numerous convulsions. Temp. and pulse very irreg. After puncture, fontanelle soft, slightly depressed, no other change. Temp., 98 to 104. Pulse, 112 to 170...........	Death; 12th day.
S. P., 10th d., 5 dr., slightly turbid, Dip. +; 10th d., 4 oz. hypo. of saline.....	Unconscious throughout. Temp., 99 to 107. Pulse, 88 to 140. Convulsions for 30 hours preceding death..........	Death; 14th day.
S. P. 4th d., single drop; slightly turbid; contained few leucocytes, Dip.—; S. P. 21st d., 6 dr.; slightly turbid; Dip. +.....................	Mental condition improved; child looks bright. Pupils normal, retraction of head and Kernig's sign well marked. Temp. high, 104, with remissions. Pulse, 120 to 140. After 19th day, failed rapidly. Totally blind, very stupid; emaciating...........................	Still in ward 25th day of disease.
S. P. 10th d., 8 dr.; clear; few leucocytes; Dip. +.	Steadily improved. Temp. low, irreg., 97 to 102. Pulse, 98 to 150. Bronchitis severe. Kernig not present at any time.	Still in ward, slight rigidity of neck persisting, 26th day.
S. P. 9th d., 14 dr.; slightly turbid; Dip. +; S. P. 14th d., 2 d.; slightly turbid, Dip. +; 14th d., 10 oz. hypo. of saline.....	Unconscious, restless, no evidence of sight or hearing at any time during stay in ward. Temp. practically normal. Pulse, irreg., 102 to 130. Numerous convulsions...........................	Death, 14th day.
S. P., 2d d., 8 dr., very turbid, Dip. +; 2d d., 5 oz. hypo of saline..........	Third day more conscious, quite irritable; head retracted to right angle; 14th day, head still retracted, mental condition quite clear. Pupils normal. Temp., remit., 100 to 102; P., 92 to 120. After 18th day diminishing rigidity; complains of headaches; shows emaciation; temp. continues......................	Still in ward 26th day.
S. P., 4th d., no fluid, puncture done with difficulty, patient resisted; 7th d., 1½ dr., slightly turbid, Dip. +................	Steady improvement. Temp., 98° to 102, with gradual decline. Pulse, 80 to 120. No rigidity after 14th day. Pupils normal throughout....................	Still in ward 25th day; T. 100. No other symptom.
S. P., 3d d., 5 dr., turbid, Dip. +; 3d d., 6 oz. hypo. of saline; 7th d., 6 oz. hypo. of saline.....	Grew worse until 7th day; unconscious; rapid emaciation; increasing rigidity. After 7th day mental condition clear; rigidity lessening. Pupils still dilated, inactive. Temp., irreg., 97 to 104. Pulse, 102 to 150. On 25th day no symptoms, except irreg. temp. and sluggish pupils......................	Still in ward 25th day.

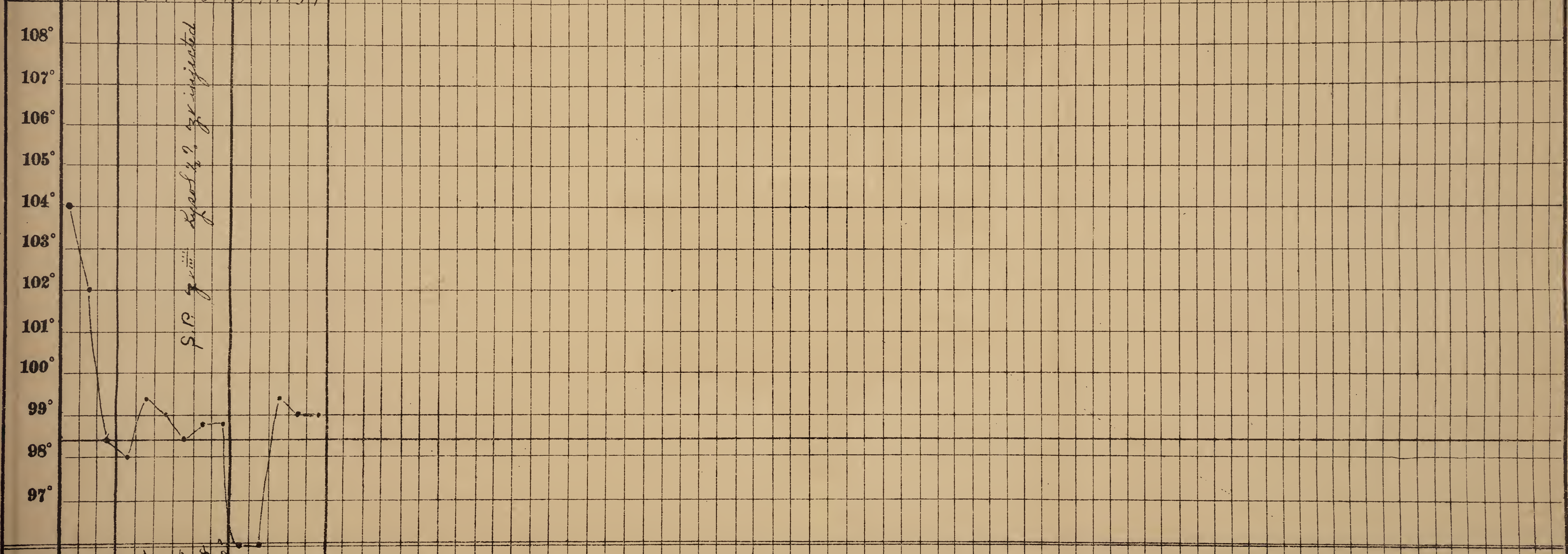
Name, Age, Address, Admitted.	Onset.	Day of Disease.	Condition on Admission.	Complication.
54. Morris G., 9 yrs., 178 Orchard st., May 16, 1904...	Pains in hands and legs; headache (occipital); chilly sensations; vomiting; convulsions 3d day....	11th	Unconscious; irritable: pupils inactive; head strongly retracted; lordoris; Kernig; emaciation; T. 102, P. 108, R. 26........	Total deafness on admission.....
55. Benj. G., 4 yrs., 173 Ludlow st., May 16, 1904..	Vomiting; headache; pain in abdomen..........	2d, 26th hr.	Conscious; stupid; pupils sluggish; slight retraction of head; Kernig; T. 100/2, P. 104, R. 32; numerous petechiae; mottled extremities..	Herpes; 4th day, conjunctivitis..
56. Lillie F., 7 yrs., 419 Cherry st., May 19, 1904..	Vomiting; headache; chill; convulsions; loss of consciousness ...	1st, 17 hr.	Unconscious; marked pallor (shock); pupils inactive; neck rigid; Kernig; T. 103/4, P. 140, feeble; R. 38, irregular; mottled extremities..	Conjunctivitis, Dip.+.........
57. Dora C., 1 yr.., 181 Clinton st., May 20, 1904..	Vomiting; convulsions	2d, 30th hr.	Semi-conscious; restless; pallor of lips and face; pupils normal; head retracted; Kernig; fontanelle tense; T. 100/4, P. 168, feeble; R. 68, irregular. Profuse petechiae and deeply mottled skin.........	Conjunctivitis, bronchitis, rhinitis; Dip.+..
58. James S., 21 yrs., 58 Gouverneur st., May 21, 1904...........	Headache; vomiting; pains in back; unconscious 1st day...	2d, 28th hr.	Coma; pupils widely dilated, inactive; nystagmus; face cyanosed; neck rigid; Kernig; T. 104/2, P. 108, R. 50. Pulse barely palpable. Respiration shallow and irregular. Profuse petechiae and large purpuric blotches on back and extremities..	None ....
59. Yetta T., 2 yrs., 124 Suffolk st., May 21, 1904..	Vomiting; T. 104; past four weeks 102° to 104 T.; 24th day became blind and unconscious...........	28th	Semi-conscious; blind; irritable; pupils dilated, inactive; neck rigid; Kernig; emaciation. T. 101/4, P. 120, R. 48........	None...........
60. Mary Tully, 3 yrs., 655 Water st., May 24, 1904	Vomiting; restlessness; stupor.....	1st, 20th hr.	Unconscious; quiet; pupils dilated, inactive; slight rigidity of neck; Kernig; T. 102, P. 90, R. 36. Few petechiae............	Conjunctivitis....

Spinal Punctures and Hypodermoclisis.	Remarks.	Result.
S. P., 11th d., few drops of pus, Dip.+; S. P., 16th d., needle clogs with fibrin, after flakes drawn out with aspirator, 7 dr. clear fluid; S. P., 18th d., 4 dr., needle clogs as before; S. P., 19th d., 5 dr., slightly turbid, flows readily	Signs of improvement about 18th day; more conscious; temp. breaking; totally deaf; rigidity lessening. Temp. continuous 101 to 103 until 18th day. Pulse, 108 to 140. After 23d day increasing stupor and emaciation; pupils dilated; low temp.	Still in ward 34th day of illness.
S. P., 2d d., 2 dr., turbid, Dip. +; 2d d., 10 oz., hypo. of saline	Condition remained about the same (rigidity increasing) until 6th day, after which steady and rapid improvement. Temp., low, irreg., 98 to 104. Pulse, 84 to 140. Temp. normal after 11th day. No symptoms after 14th day.	Recovery complete. Discharged 15th day.
S. P., 1st d., on admis. 4 dr., turbid, Dip.+; 1st d., on admis. 10 oz. hypo. of saline; 2d d., 10 oz. hypo. of saline; S. P., 5th d., 4 dr., turbid, Dip.+	Eight hours after admission: semi-conscious, restless; color good; pulse fair. On 2d day mental condition clear in A. M., active delirium in P. M.; injection of saline repeated. On 3d day, quiet; mental condition clear. On 5th day, somewhat irrational; pupils inactive (S. P., 4 dr.) Since 5th day mental condition has remained clear; the pupils reacting fairly well: hyperaesthesia has been marked. Temp., course, high, irreg., 98 to 104. Pulse, 84 to 148	Still in ward 19th day.
S. P., 2d d. on admis., 6 dr., turbid, Dip. +; 2d d. on admis., 4 oz. hypo. of saline	Ten hours after admission more quiet; pulse and color much better; more rigidity. Has continued stupid and irritable. Temp. high, irregular, 99 to 104. Pulse 120 to 168. After 12th day no symptoms except irregular temp.	Still in ward 19th day.
S. P., 2d d. on admis., 6 dr., very turbid; coagulates, many leucocyts, Dip. +; 2 d. on admis., 1 pint hypo. of saline; 2d d., 1 pint hypo. of saline.	Ten hours later color good; pulse fair, quite restless; pupils small; still unconscious. Temp. 99 to 104 24 hours after admission again in profound coma. Temp. 104. Pulse 130. Resp. slow and irregular	Death; 3d day
S. P., 28th d., 5 dr., slg. turbid, Dip. +; 28th d., 6 oz. hypo. of saline; 35th d., 5 dr., slg. turbid	Remains blind, semi-conscious and irritable. Temp. irregular, 99 to 103. Pulse 100 to 144	Still in ward, 45th day of illness.
S. P., 1st d., on admis., 4 dr., turbid, Dip. +; on admis. 10 oz. hypo. of saline	On 2d day semi-conscious, more rigidity of neck and extremities. On 3d day, temp. 104, pulse 120, colon irrigation at 90 degrees was given. Temp. dropped to 98, pulse 72. Death few moments later. Temp. was irreg., 100 to 104, pulse 90 to 140	Death; 3d day.

AGE 12 YEARS. MALIGNANT TYPE.

Name Moses Gauber Admitted Mar 28 19 04 Division, Ward M. M. Temperature Chart No. 1

Date	28			29						30				
Day of Disease	2			3						4				
Hour	2	5	9	1	5	9	1	5	9	5	9	1	5	9



S.P. ℥ viii Lysol ½ %. ℥x injected

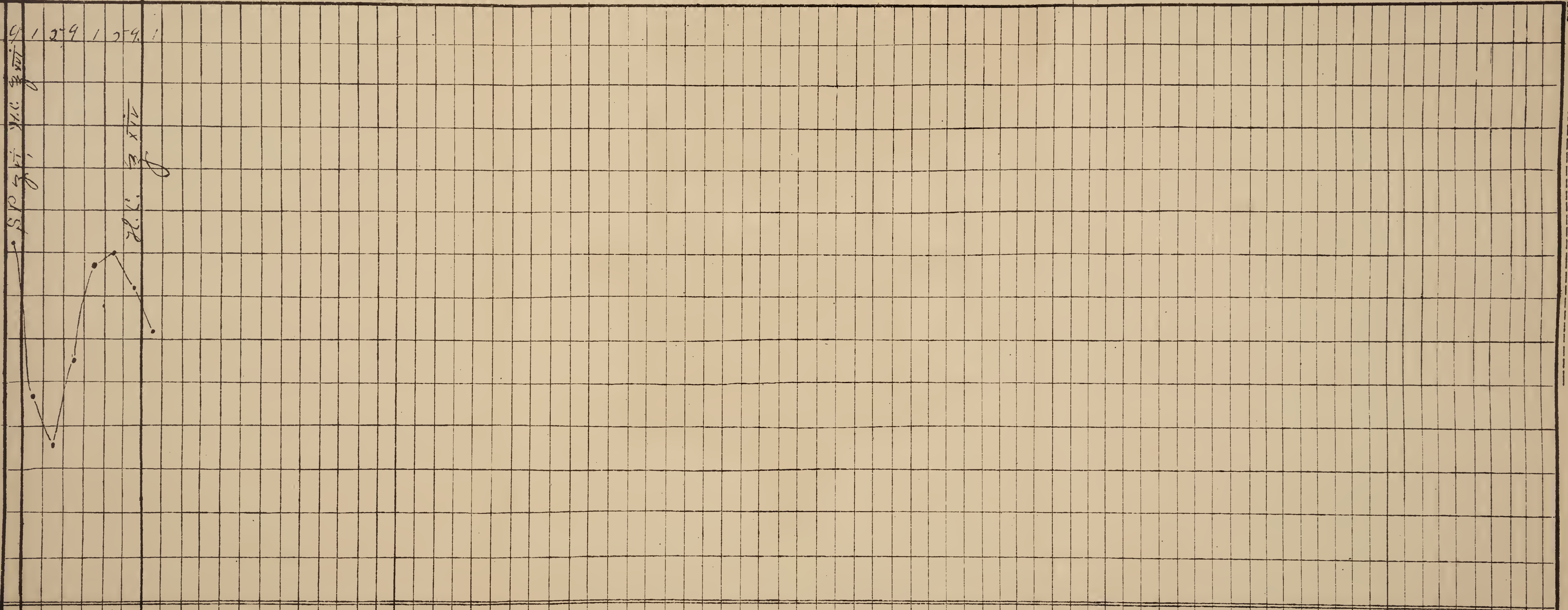
Pulse	72	72	60	?	88	86	138	128	140?	?	?	?	?	?
Resp.	32	28	24	30	28	26	36	30	?	?	60	36	44	38
Total Urine														
Total Def.														

Diagnosis

Serial No.

AGE 21 YEARS. MALIGNANT TYPE

Name James Smith Admitted May 21 19 4 Division, Ward M. M. Temperature Chart No. 1



Date	21	22						23
Day of Disease	1	2						3
Hour	9	1	5	9	1	5	9	1
Pulse	108	112	108	106	108	128	130	116
Resp.	50	34	34	24	28	28	30	40
Total Urine								
Total Def.								

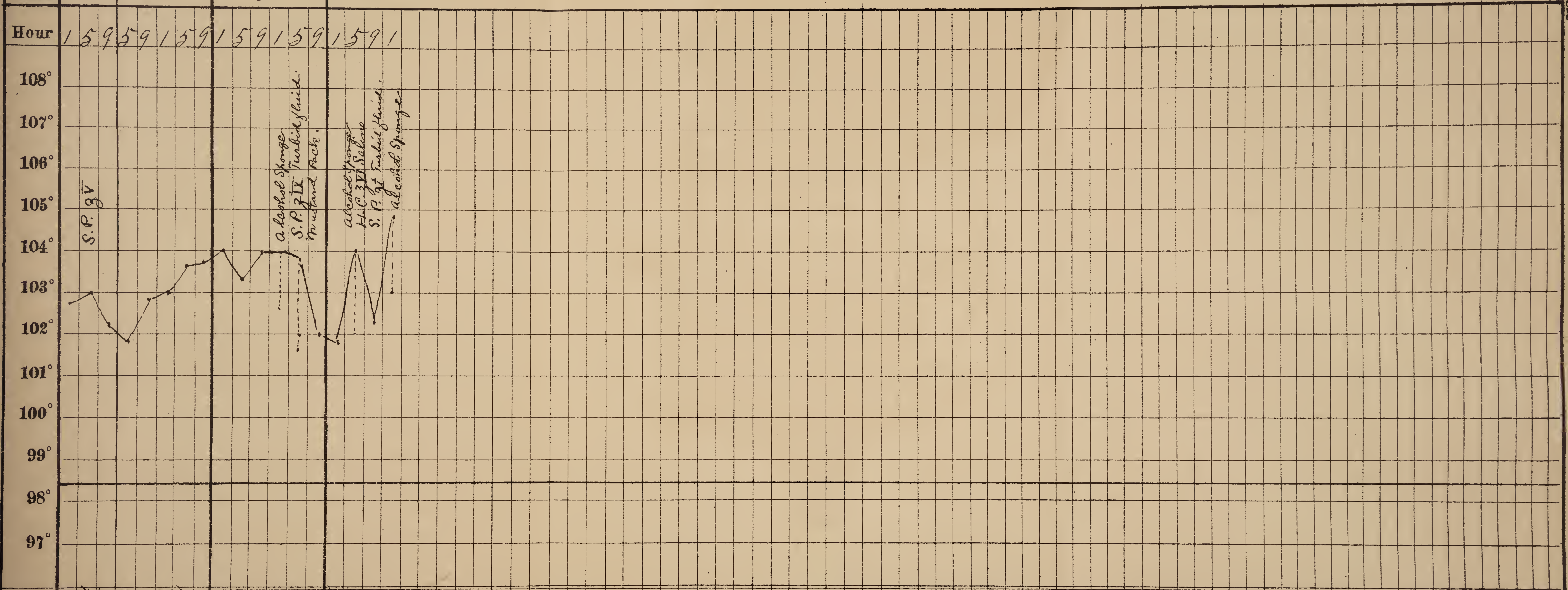
Diagnosis

Serial No.

*35-AGE 10 MONTHS. TOXAEMIA.

Name *Edward D.* Admitted *May 4* 19*14* Division, Ward *Ch. Med.* Temperature Chart No. *1*

Date	May 4	5	6	7
Day of Disease	1	2	3	4
Total Urine	ı	ı ı ı	ı ı ı	
Total Def.	ı	ı ı ı	ı ı ı	

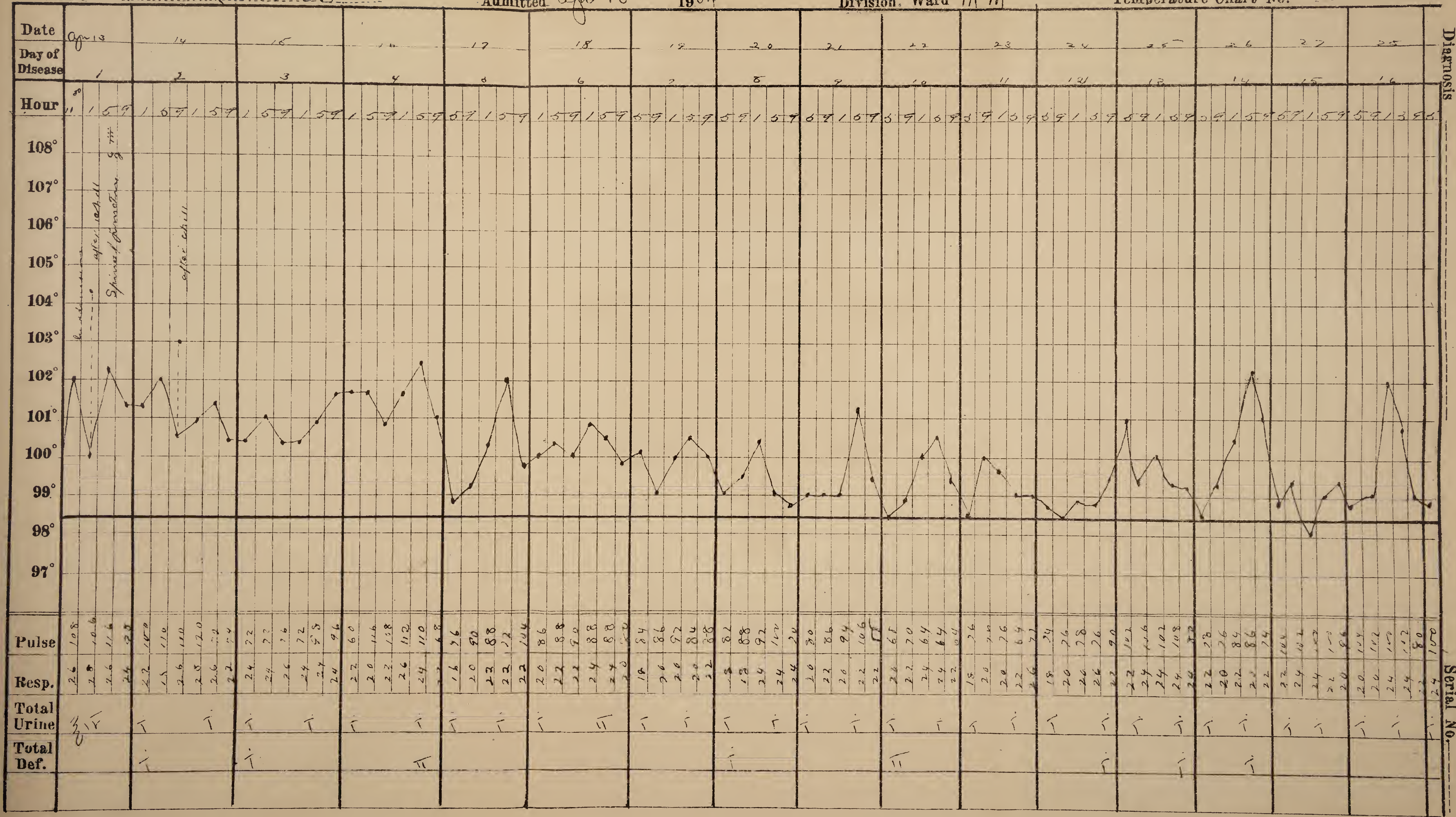


Hour	1	5	9	5	9	1	5	9	1	5	9	1	5	9	1	5	9
Pulse	140	144	138	142	140	142	144	146	140	140	160	148	152	150	144	154	140
Resp.	50	52	54	50	48	48	50	52	54	52	50	52	48	50	42	52	48

Serial No.

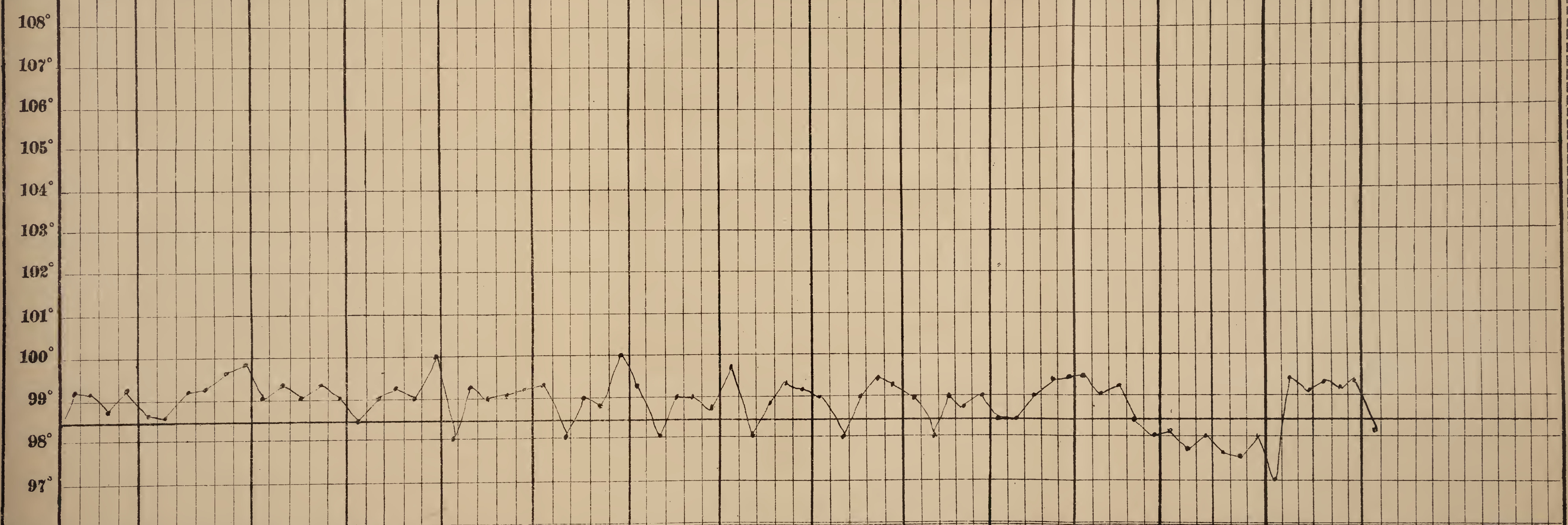
ABORTIVE TYPE WITH SLIGHT RECRUDESCENCE.

Name Frank Lewis Admitted Apr 13 1904 Division. Ward M M Temperature Chart No. 1.



Name Frank Lewis Admitted Apr 13 19 04 Division, Ward M. D. Temperature Chart No. 2

Date	Day of Disease	Hour	Pulse	Resp.
29	17	9 1 5 9	82 88 86 100	24 26 24 24
30	18	1 5 9 1 5 9	100 94 102 104 106 96	24 22 26 24 26 22
May 2	19	5 9 1 5 9	96 96 78 60 80	24 24 22 24 24
3	20	5 9 1 5 7	86 120 116 118 60	23 24 22 24 24
4	21	5 9 1 5 9	60 90 98 94 96	22 22 24 22 22
5	22	5 9 1 5 9	88 70 96 92 94	20 22 20 22 22
6	23	5 9 1 5 9	88 94 86 90 96	20 22 20 26 24
7	24	5 9 1 5 7	80 116 106 100 84	20 24 22 24 20
8	25	5 9 1 5 9	90 84 90 96 82	21 24 20 24 20
9	26	5 7 1 5 9	90 96 94 96 90	18 20 22 20 18
10	27	5 9 1 5 9	100 94 86 96 92	20 22 20 24 22
11	28	5 9 1 5 9	100 96 96 94 92	20 24 22 20 22
12	29	9 3 9	90 90 84	20 20 22
13	30	9 3 9	86 80 72	20 22 24
14	31	9 3 9	106 100 90	22 22 20
15	32	9 3 4 9	92 84 96	20 20 28



Total Urine

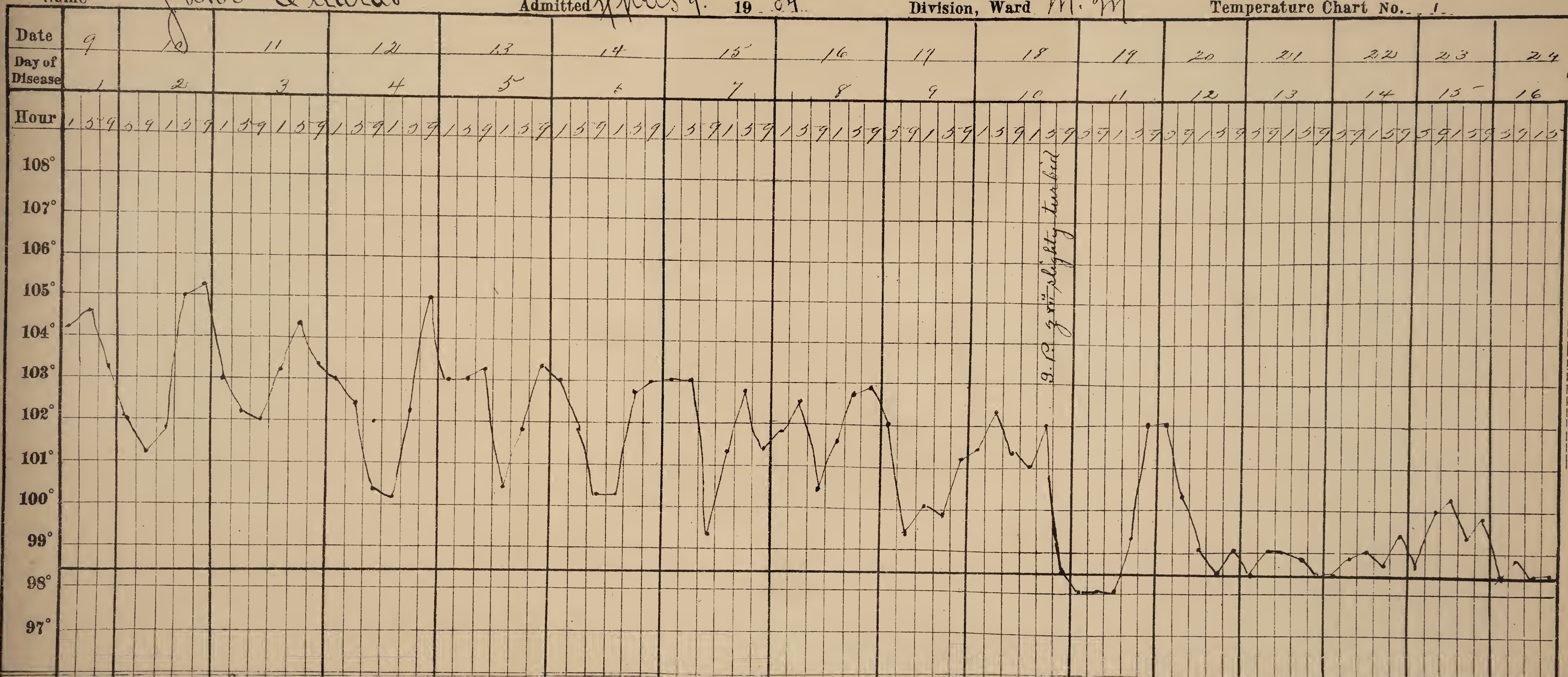
Total Def.

Diagnosis

Serial No.

*18-AGE 30 YEARS. MILD TYPE.

Name Jake Euardo Admitted April 9. 19 04. Division, Ward M. M Temperature Chart No. 1.



Pulse	Resp.
76	24
88	24
88	22
62	24
84	22
88	24
86	24
74	20
70	24
68	16
60	18
62	16
64	20
72	22
72	18
76	20
72	18
66	24
76	24
76	24
76	24
74	24
60	22
70	24
72	22
92	22
76	20
70	20
72	22
86	26
68	24
60	22
68	22
72	22
60	22
72	22
80	28
70	28
70	20
68	20
70	22
76	24
76	24
100	24
66	18
72	20
80	20
86	22
84	22
70	18
68	18
72	20
84	24
78	24
84	24
88	20
76	20
80	20
76	20
76	20
90	18
76	22
84	24
80	24
72	22
86	22
72	24
78	24
80	24
88	24
90	22
76	21
72	26
88	24
88	22
80	20
86	22
88	22
90	22
82	22
70	22
84	20
72	24
78	24

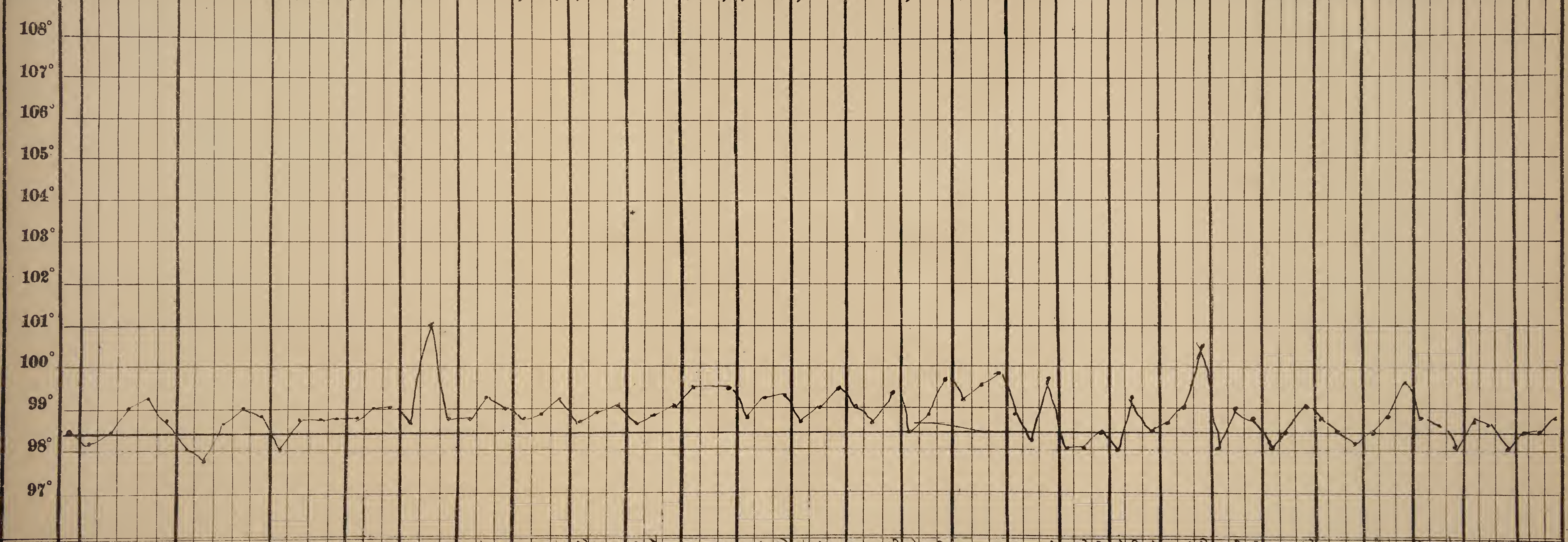
Total Urine

Total Def.

Serial No.

Name Jake Edwards Admitted April 9 19 04 Division, Ward M.M. Temperature Chart No. #

Diagnosis



Date	Day of Disease	Hour	Pulse	Resp.
		9	72	18
25	17	5 9 1 5 9	72 88 86 88 66	18 24 24 24 18
26	18	5 9 1 5 9	72 72 80 88 64	18 20 24 24 18
27	19	5 9 3 9	64 80 88 72	18 20 24 20
28	20	9 3 9	80 86 62	20 24 22
29	21	9 3 9	72 76 80	24 26 22
30	22	9 3 9	84 68 60	24 24 22
May	23	9 3 9	66 70 78	20 20 22
2	24	9 3 9	72 68 60	22 20 20
3	25	9 3 9	70 74 60	20 20 18
4	26	9 3 9	72	18
5	27	9 3 9	76 78 72	22 20 20
6	28	9 3 9	84 82 88	24 24 22
7	29	9 3 9	96 80 72	28 24 18
8	30	9 3 9	92 90 90	24 24 22
9	31	9 3 9	80 76 80	20 22 20
10	32	9 3 9	100 88 72	24 20 18
11	33	9 3 9	96 92 110	20 24 24
12	34	9 3 9	92 100 92	20 24 22
13	35	9 3 9	96 92 100	20 24 24
14	36	9 3 9	92 100 92	20 24 22
15	37	9 3 9	86 80 72	20 22 24
16	38	9 3 9	76 80	20 22
17	39	9 3 9	92 84	22 20
18	40	9 3 9	100 98 88	22 20 20
19	41	9 3 9	80 64 84	24 20 24
20	42	9 3 9	90 88 74	22 24 22

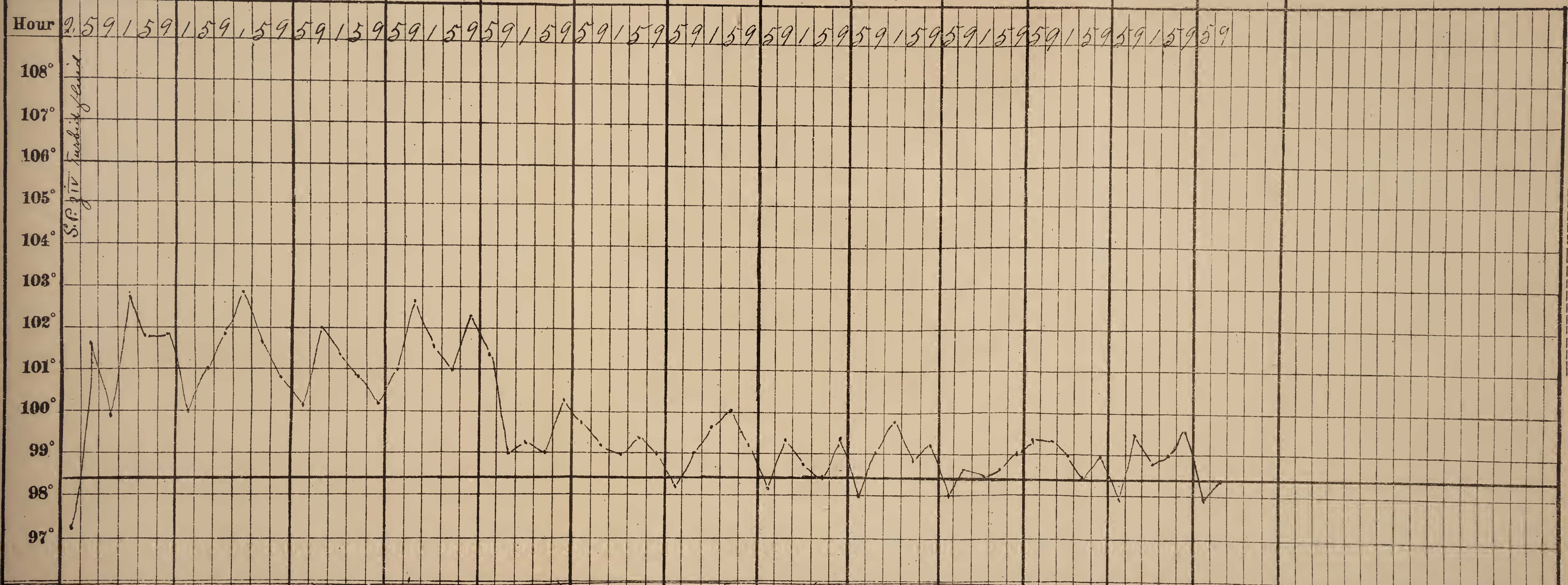
Total Urine

Total Def.

Serial No.

* 26-AGE 6 YEARS. ABORTIVE TYPE.

Name Dolly K. Admitted Apr 22 1904 Division, Ward Ch. Med. Temperature Chart No. 7



S.C. ℥iv turbid fluid

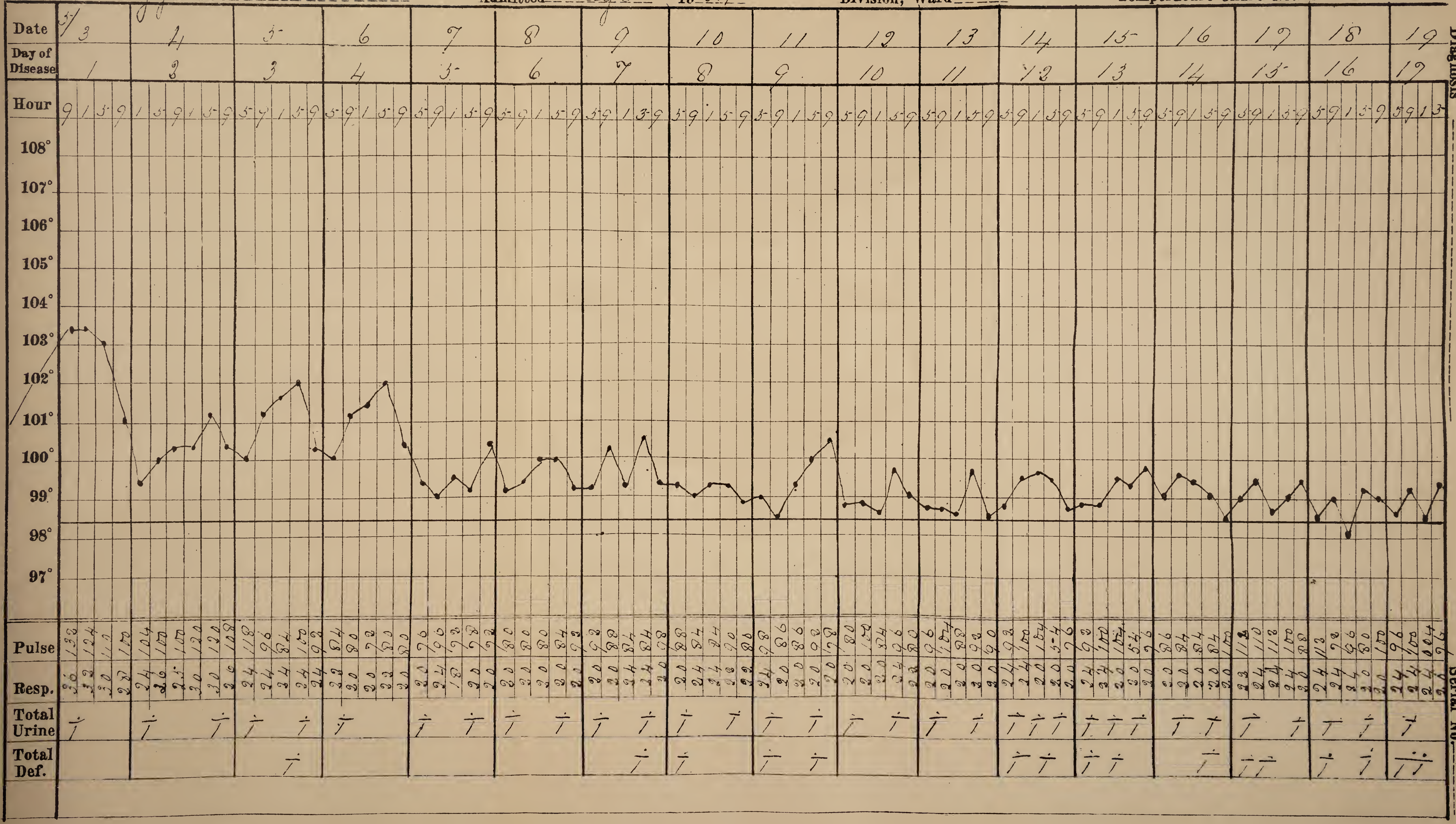
Date	Day of Disease	Hour	Pulse	Resp.	Total Urine	Total Def.
22	1	2 5 9 1 5 9	92 110 106 112 116 112	24 28 24 24 24 24	i i	
23	2	1 5 9 1 5 9	108 90 98 104 106 102	24 24 24 24 24 28	i i i	i
24	3	5 9 1 5 9	100 104 100 98 90	26 24 24 24 24	i i i	
25	4	5 9 1 5 9	98 102 108 102 110	26 24 24 24 26	i i	
26	5	5 9 1 5 9	100 94 98 102	24 24 24 22	i i i	i i
27	6	5 9 1 5 9	96 96 92 98 94	26 24 26 26 24	i i i	i
28	7	5 9 1 5 9	90 98 104 108 96	26 24 24 24 26	i i i	i
29	8	5 9 1 5 9	94 96 100 108 102	24 24 24 24 24	i i i	
30	9	5 9 1 5 9	96 102 100 106 100	26 24 26 24 26	i i	i i
May 1	10	5 9 1 5 9	94 120 100 102 100	24 24 24 26 24	i i i	
2	11	5 9 1 5 9	98 100 96 94 98	26 24 24 22 26	i i i	i
3	12	5 9 1 5 9	94 98 96 98 102	24 24 24 24 26	i i i	i
4	13	5 9	92 100	24 24	i	

Diagnosis

Serial No.

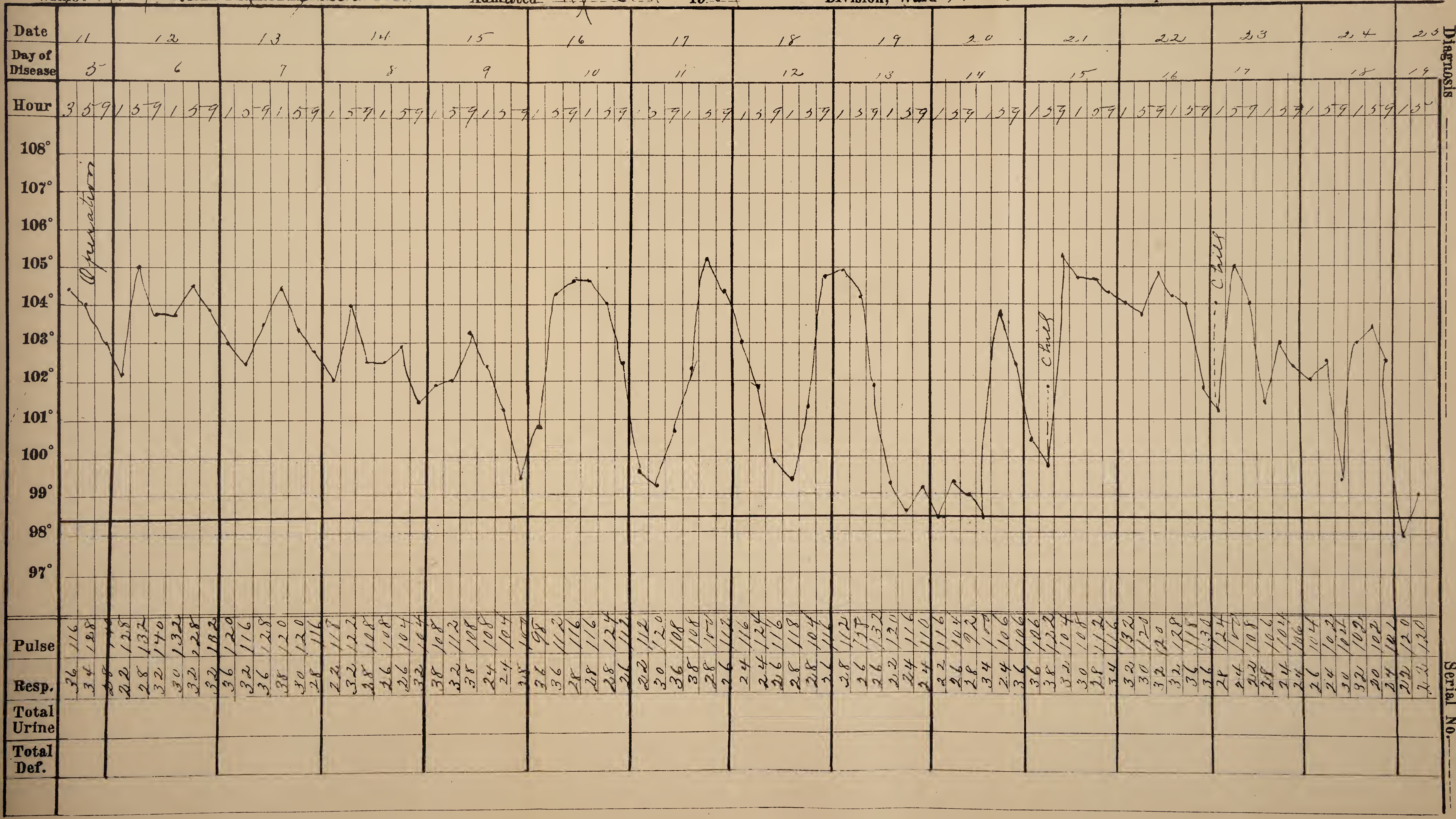
#38-AGE 16 YEARS. ABORTIVE TYPE

Name Maggie Plain Admitted May 3rd 19 04 Division, Ward F. M. Temperature Chart No. I

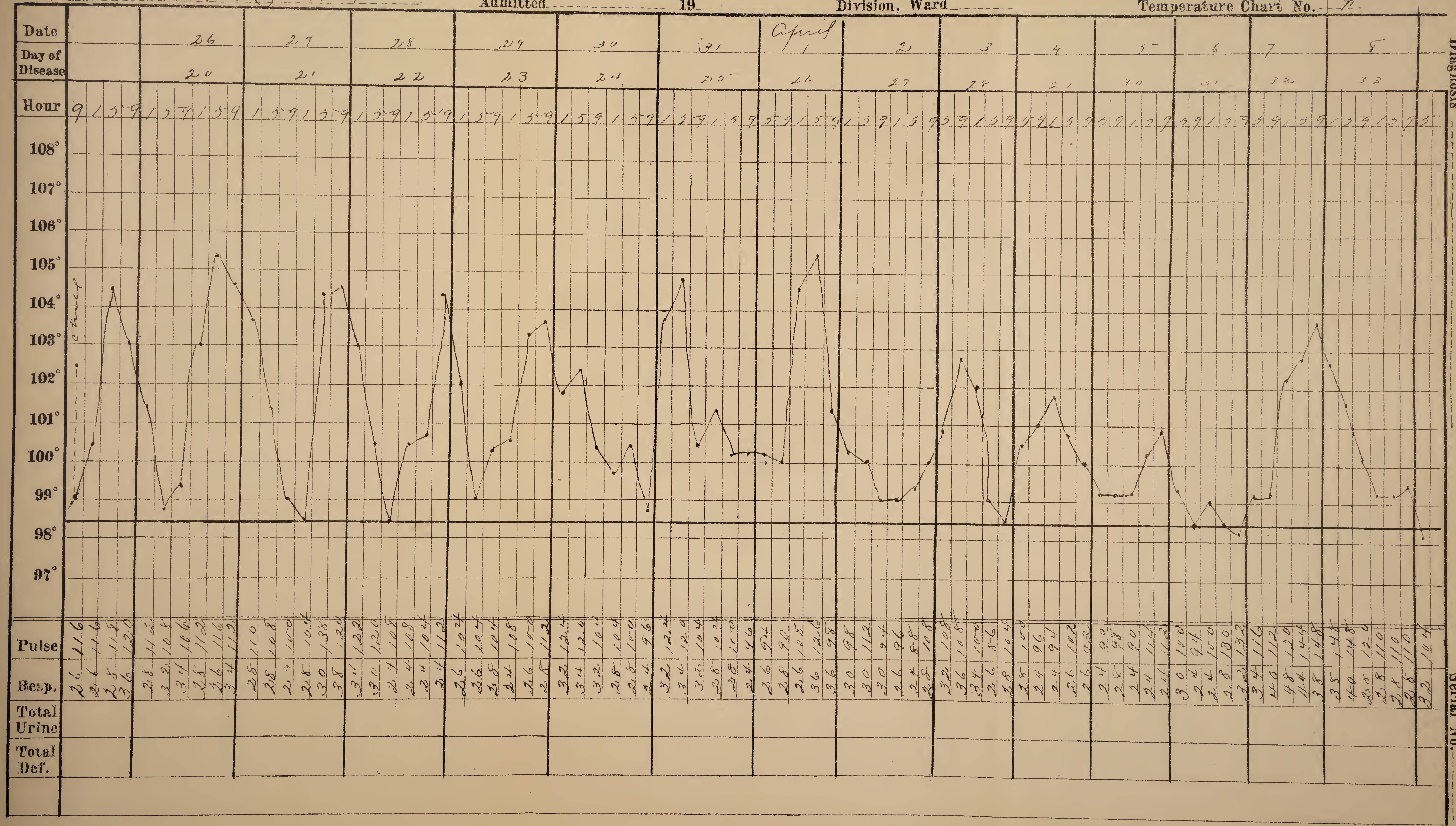


#3-AGE 16 YEARS. INTERMITTENT TYPE.

Name Sarah Lechtar Admitted March 11 1907 Division, Ward Xem Temperature Chart No. 1



Name Sarah Lichtas Admitted ______ 19__ Division, Ward ______ Temperature Chart No. ii

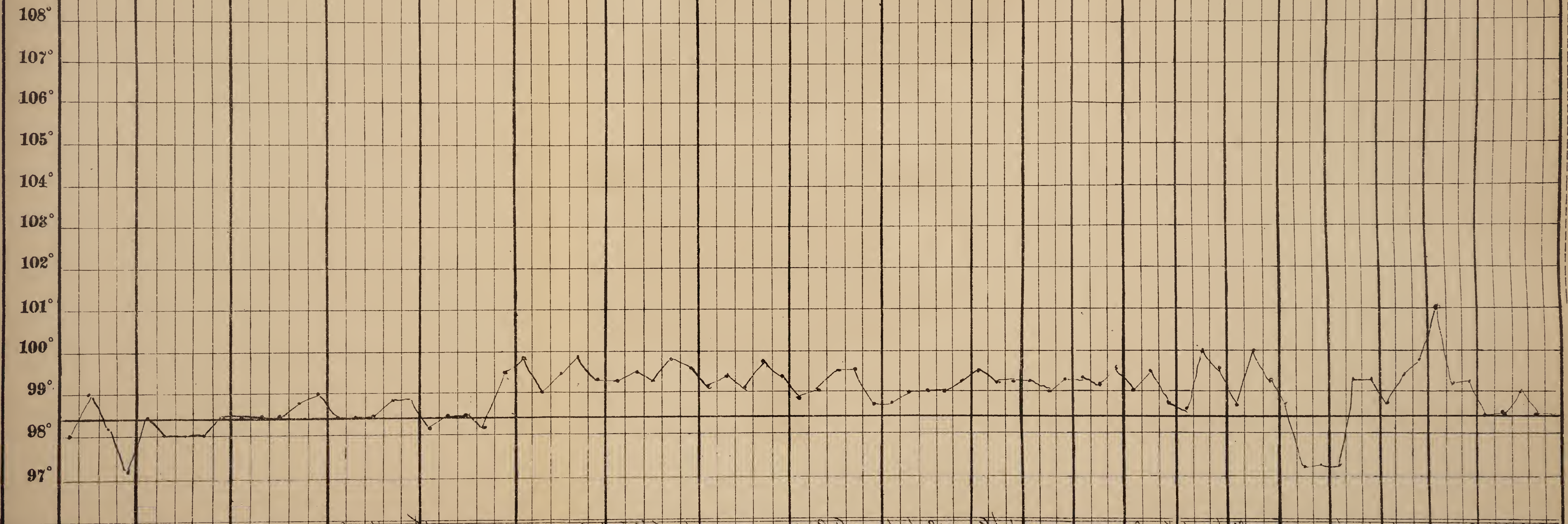


Date	Day of Disease	Pulse	Resp.
		116 116 118 120	26 26 28 36
26	20	118 108 116 112 116 112	28 32 34 28 36 34
27	21	110 108 100 104 138 120	28 28 24 28 30 38
28	22	122 120 108 108 104 112	34 30 24 24 24 24
29	23	104 104 104 108 100 112	26 26 28 24 26 28
30	24	124 120 104 104 100 96	32 34 32 28 28 24
31	25	124 120 104 102 100 96	32 34 32 28 28 24
April 1	26	94 90 108 126 98	26 28 26 36 36
2	27	98 112 94 96 88 108	30 30 30 26 28 28
3	28	108 108 100 86 104	32 36 34 26 28
4	29	100 96 94 112 102	28 24 24 26 26
5	30	90 98 90 114 112	24 26 24 24 24
6	31	100 94 100 130 132	30 24 28 28 32
7	32	116 112 120 144 148	34 40 48 44 38
8	33	148 148 120 110 110 110	38 40 30 28 28 28
		104	32

Name Sarah Lichtar Admitted May 11 19 04 Division, Ward Temperature Chart No. III

Diagnosis

Date	Day of Disease	Hour	Pulse	Resp.
9	34	9	96	48
		1	112	28
		5	116	32
		9	108	30
10	35	5	102	18
		9	112	20
		1	112	24
		5	116	26
		9	108	32
11	36	5	112	28
		9	112	28
		1	114	28
		5	96	28
		9	132	24
12	37	5	112	22
		9	112	24
		1	124	28
		5	112	32
		9	128	30
13	38	5	108	28
		9	108	28
		1	106	24
		5	106	24
		9	108	24
14	39	5	92	26
		9	112	28
		1	110	28
		5	112	28
		9	112	28
15	40	5	112	28
		9	122	26
		1	124	28
		5	100	32
		9	128	30
16	41	5	118	26
		9	120	28
		1	96	28
		5	96	28
		9	112	28
17	42	5	108	28
		9	120	24
		1	100	24
		5	100	24
		9	120	26
18	43	5	108	24
		9	108	24
		1	100	28
		5	116	28
		9	100	24
19	44	9	120	30
		3	108	30
		9	118	30
20	45	9	96	24
		3	100	24
		9	100	22
21	46	9	96	24
		3	100	24
		9	90	22
22	47	9	100	24
		3	118	24
		9	124	24
23	48	9	108	24
		3	110	26
		9	108	24
24	49	9	106	24
		3	140	24
		9	100	24
25	50	9	104	24
		3	96	24
		9	116	24
26	51	9	108	24
		3	96	24
		9	108	24
27	52	9	132	24
		3	132	24
		9	104	24
28	53	9	132	22
		3	120	24
		9	132	24
29	54	1	92	22
		5	120	20
		9	106	20
		1	106	22
		5	96	22



Total Urine

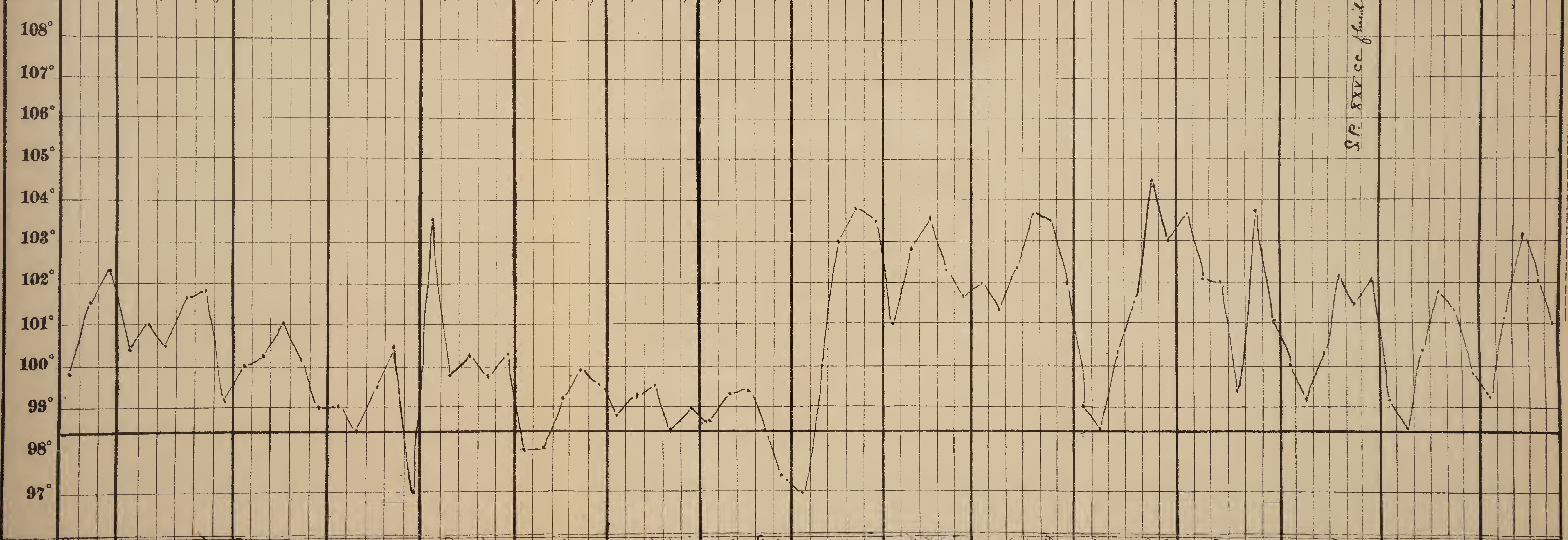
Total Def.

Serial No.

*5-AGE 4 YEARS. DEATH 42ND DAY. GREAT EMACIATION.

Name Willie L. Admitted Mar 14 1904 Division, Ward Ch. Med. Temperature Chart No. 1

Date	Mar. 14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29
Day of Disease	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Hour	1 5 9	1 5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	5 9 1 5 9	1 5 9 1 5 9	1 5 9 1 5 9	1 5 9 1 5 9	1 5 9 1 5 9	1 5 9 1 5 9	1 5 9 1 5
Pulse	108 112 116	112 120 120 124 108	108 120 108 120 100	96 104 118 136 96	100 120 122 144 108	92 96 132 100	92 96 104 104 100	88 112 120 108 84	88 112 120 140 112	100 124 134 108 120	96 120 108 124 124 100	112 108 84 108 108 108	100 100 108 112 130 100	96 100 110 120 100 100	92 88 120 100 100 100	96 88 96 104 108
Resp.	32 30 36	40 32 30 26 24	24 24 28 24 24	24 24 20 32 24	24 26 28 26 24	24 24 26 24	24 24 26 24 20	20 28 18 24 20	20 24 24 26 28	28 28 32 24 24	20 24 24 28 26 24	20 24 24 24 24 20	24 20 24 26 20 24	24 24 24 24 24 24	20 20 24 24 24 24	24 20 26 24 26
Total Urine	i i	i i i	i i i	i i i	i i i	i i i	i i	i i	i i i	i i i	i i i	i i i	i i i	i i i	i i i	i i
Total Def.	i	i i i i	i	i	i i	i i	i i	i i	i	i		i i	i i	i	i	i

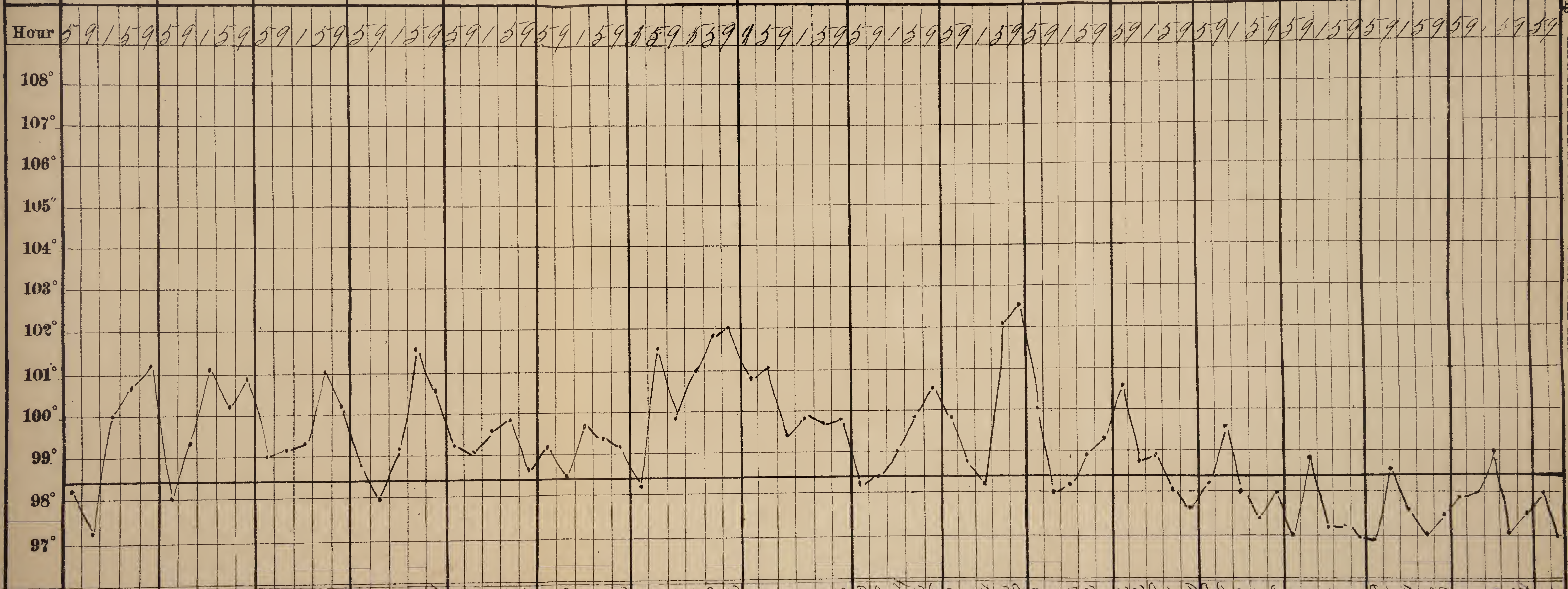


Diagnosis

Serial No.

Name *Willie L.* Admitted *Mar. 14* 19*04* Division, Ward *Ch. Med.* Temperature Chart No. *II* —

Diagnosis



Date	Day of Disease	Pulse	Resp.	Total Urine	Total Def.
Mar. 30	17	100 120 108 120 100	20 18 20 24 20	i i i	i i
31	18	80 120 108 100 120	20 24 24 24 24	i i i	i i
Apr. 1	19	84 120 124 132 108	20 24 24 30 20	i i i	i i
2	20	112 104 120 120 124	24 26 18 24 24	i i i	i
3	21	120 120 110 104 104	26 24 24 24 24	i i i	
4	22	116 120 112 118 102	28 26 24 24 24	i i i	
5	23	94 120 120 100 118 122	24 22 26 22 24 24	i i i	i i i
6	24	104 116 102 112 100 106	26 24 24 26 26 24	i i i	
7	25	100 98 124 112 108	24 26 26 24 26	i i i	
8	26	100 98 124 112 108	24 26 26 24 26	i i i	
9	27	100 116 112 102 110	24 28 26 26 24	i i i	i
10	28	102 92 98 94 120	24 24 24 26 28	i i i	i
11	29	98 108 100 96 98	26 26 24 24 26	i i i	
12	30	90 96 102 94 90	28 26 24 26 24	i i i	
13	31	108 114 104 96 100	28 26 24 26 28	i i i	
14	32	96 100 92 96 94 96 84	26 26 20 24 26 26 26	i i i i	i i

Serial No.

Name Willie L. **Admitted** Mar. 14 **19** 04 **Division, Ward** Ch. Med. **Temperature Chart No.** iii

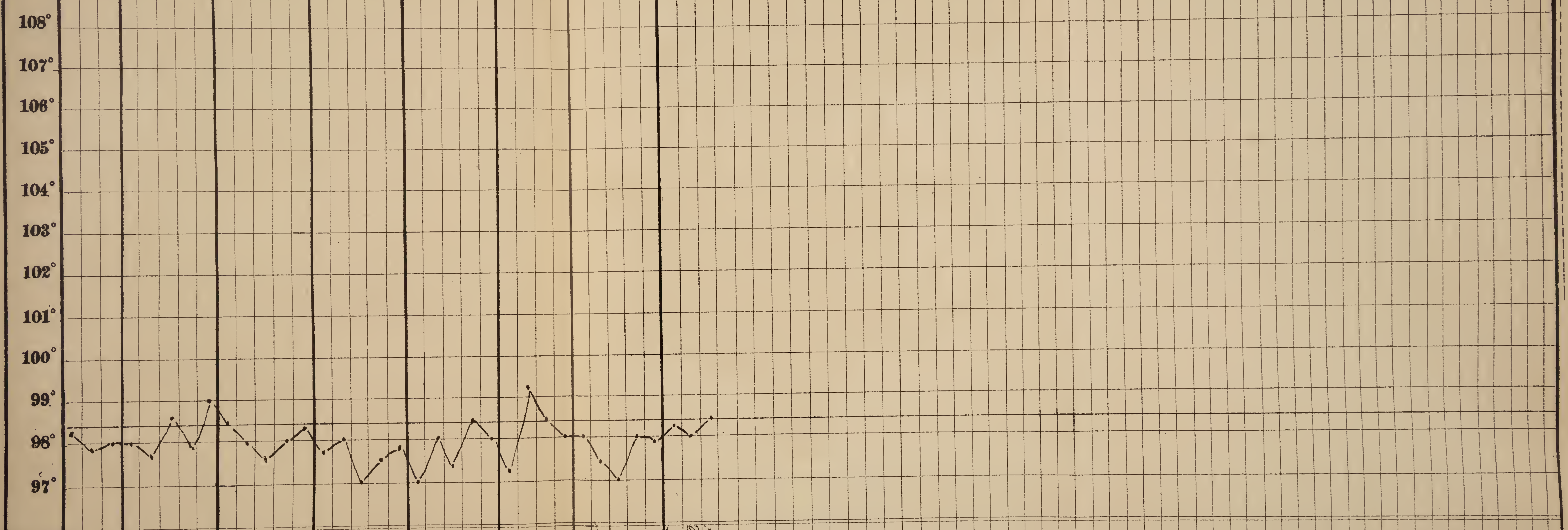
Diagnosis

Serial No.

Date	Apr. 15			16					17				
Day of Disease	33			34					35				
Hour	1	5	9	5	9	1	5	9	5	9	1	5	9
Pulse	92	96	94	88	90	84	94	92	86	84	90	100	94
Resp.	24	24	22	26	24	24	24	26	24	28	26	30	28
Total Urine		i			i	i	i			i	i	i	
Total Def													

Date	18					19					20			
Day of Disease	36					37					38			
Hour	5	9	1	5	9	5	9	1	5	9	5	1	5	9
Pulse	96	98	92	96	88	92	98	90	100	94	90	96	92	98
Resp.	26	26	24	24	22	26	24	24	24	26	32	28	28	26
Total Urine		i		i	i		i		i	i		i	i	i
Total Def								i						

Date	21					22		
Day of Disease	39					40		
Hour	5	9	1	5	9	5	9	1
Pulse	90	96	96	96	100	108	102	104
Resp.	24	24	24	24	24	24	24	24
Total Urine		i	i		i		i	i
Total Def								



REPORT OF FORTY-SIX CASES OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

W. K. Draper, M. D.

Forty-six cases of epidemic cerebro-spinal meningitis were treated in the wards of the First, Second and Third Medical Divisions of Bellevue Hospital between the 1st of February and the 30th of April, 1904. A number of cases have been admitted since the latter date, but of these the statistics are not included in the following report.

By the courtesy of the attending physicians on duty in the wards of the Second and Third Medical Divisions during the months mentioned, the histories of the cases treated by them have been placed at the disposal of the writer and are included in this report.

Of the forty-six cases Numbers I. to XXIX., inclusive, were treated in the First Medical Division; XXX. to XXXVII., inclusive, and Case Number XLV., were cared for in the Second Medical Division; Cases XXXVIII. to XLIV., inclusive, were in the service of the Third Medical Division; and Case XLVI., a physician, a former member of the House Staff, was under the care of Dr. W. Gilman Thompson, assisted by Dr. E. P. Warren, of the First Medical Division.

In a number of the cases it was impossible to obtain satisfactory histories on account of the mental condition of the patients. Complete records, also, were not kept in all instances on account of the extremely active service and the fact that at times the Interne Staff was crippled by illness. Of the series twenty-nine cases occurred in males and seventeen in females. Twenty-six were adults and twenty were children. The total mortality was 73.9 per cent. Of the children (those under 15 years of age) fifteen died and five recovered, a mortality of 75 per cent. Of the adults nineteen died and seven recovered, which represents a mortality of slightly more than 73 per cent. There were thirteen cases treated by the method of Lysol injections into the sub-dural space. Of these eight died and five recovered, a mortality of 61.5 per cent., rather less than that of the cases treated by the expectant plan. In twenty-eight of the cases the onset was acute; in thirteen, gradual, and in five, the mode of onset was not noted. Four of the cases terminated fatally within forty-eight hours after the first symptom developed. The majority of the cases came from tenements located on the East Side of the city, between Thirtieth and Fiftieth streets, but this was undoubtedly due to the fact that Bellevue Hospital draws most of its patients from this locality. Other hospitals in other parts of the city have had a large number of similar cases; the epidemic, therefore, may fairly be described as widely distributed.

The following table gives these details more minutely:

CASE NO.	SEX.	AGE.	ONSET.	DURATION.	TREATMENT.	RESULT.
I	M.	7 years	Sudden	6 days; in hospital 20 hours		D.
II	M.	4½ years	Acute	30 hours	Ice, Chloral.	D.
III	M.	6 months	Gradual.	4 weeks	General.	D.
IV	F.	2 years	Acute	6 weeks	Ice, General	D.
V	F.	6 years	“	7½ weeks	“	C.
VI	M.	17 years	“	5 weeks	“	D.
VII	M.	6 years	“	46 hours	“	D.
VIII	M.	6 years	Gradual.	4 days	Lysol, Ice	D.
IX	F.	47 years	Acute	3–4 weeks	Ice, General	D.
X	F.	20 months	Gradual.	6 weeks	“	D.
XI	M.	25 years	Acute	28 hours	Chloral	D.
XII	M.	39 years	“	5 days	Lysol, Ice	D.
XIII	M.	15 years	“	7 weeks	“	C.
XIV	F.	8 years	“	5 days	“	D.
XV	F.	7 years	Gradual.	8 weeks	“	C.
XVI	F.	5 years	Acute	9 weeks	Lysol	D.
XVII	F.	17 years	“	5 days	“	D.
XVIII	F.	9 years	“	7 weeks	“	C.
XIX	F.	31 years	“	6 weeks	“	D.
XX	M.	2 years	“	20 hours	Stimulants	D.
XXI	F.	20 months	Gradual.	6 weeks	Lysol	D.
XXII	M.	30 years	Acute	3 weeks	Ice, General.	D.
XXIII	M.	5 years	“	5 days	“	D.

CASE NO.	SEX.	AGE.	ONSET.	DURATION.	TREATMENT.	RESULT.
XXIV....	M.	13 years..........	Gradual.	5 days	Ice, General.....	D.
XXV...	M.	5 years..........	Acute ...	4½ weeks...	"	C.
XXVI....	M.	24 years..........	" ...	4 days......	General	D.
XXVII....	M.	2 years 8 months..	" ...	8 weeks	Ice, General.....	D.
XXVIII....	M.	5 years..........	" ...	6 weeks	"	C.
XXIX....	F.	12 years..........	Gradual.	11 days......	Protargol	D.
XXX....	M.	32 years..........	"	10 days	Ice, General....	D.
XXXI....	F.	43 years..........	Acute ...	8 weeks	Lysol, Ice.......	C.
XXXII....	M.	27 years..........	" ...	4 weeks	Ice, General.....	C.
XXXIII....	M.	24 years..........	Gradual.	10 weeks	General	C.
XXXIV....	F.	24 years..........	"	4 weeks	Cold, General. ..	D.
XXXV....	M.	18 years..........	"	5 weeks	General.........	C.
XXXVI....	M.	48 years..........	Acute ...	3 days......	Ice, General.	D.
XXXVII....	M.	21 years..........	" ...	6 days......	General	D.
XXXVIII....	M.	17 years..........	" ...	4 days......	Ice, General.....	D.
XXXIX....	M.	17 years..........	" ...	5 days.....	Lysol	D.
XL....	F.	65 years..........	....	7 days	....	D.
XLI....	F.	31 years..........	....	....	Ergot	D.
XLII....	F.	47 years..........	....	....	"	D.
XLIII....	M.	33 years..........	Gradual.	8 days	....	D.
XLIV....	M.	38 years..........	Acute...	....	Lysol, Morphine.	C.
XLV....	M.	22 years..........	Gradual.	4 days......	....	D.
XLVI....	M.	....	"	9 weeks	Ice, Morphine, etc.	C.

There were several interesting instances where more than one case occurred in the same family. Cases II. and IV. were brother and sister; both were healthy children and had been well until seized with this illness. The boy was attacked suddenly in the night and within thirty-six hours the girl was stricken. It could not be learned that there were any other cases in the same tenement. Case V.—A brother of patient died two days before her admission to hospital, after an illness of thirty-six hours with symptoms of vomiting and pain in stomach. A sister was admitted to hospital in May with headache, which had persisted for a month, fever and leucocytosis: these symptoms all subsided and the patient was discharged cured. Another sister was admitted to hospital early in May with headache, vomiting and convulsions, and died in the hospital. Lumbar puncture was done in both of these cases, but the cultures in each were negative. From the symptoms, however, the cases were considered to be epidemic cerebro-spinal meningitis.

Cases XII., XV. and XVI.—Father and two daughters. The elder of the two daughters was first taken ill, ten days before she was admitted to the hospital; the onset, which was gradual, began with headache, increasing in severity, followed by vomiting. The younger sister, who slept in the same bed with the elder, was taken ill four days after the latter. Her attack began suddenly with a convulsion and coma. The father was seized one week after the elder daughter's illness began, and three days after the second daughter became sick. He was the first of the three brought to the hospital, the children following in three days. Of these three cases the father and younger daughter died, the father after an illness of five days, and the daughter after a protracted sickness of nine weeks. These three cases were all treated with Lysol injections and the diplococcus intracellularis meningitidis was demonstrated in the smears and cultures obtained by lumbar puncture in each case.

Cases XVII. and XVIII.—Two sisters, 17 and 9 years of age, respectively, the eldest and youngest of a family of five children who, with the parents, lived in four rooms. Six days before admission, the youngest child vomited but was apparently perfectly well after this until the day before she came to the hospital. This was the day following that on which the elder sister had been admitted. She, the elder, was attacked acutely twenty-four hours before admission, and was brought to the hospital in a semi-comatose condition. Of the two sisters the elder died five days after the onset and the younger recovered after an illness of many weeks. Both of these patients were treated with the Lysol injections. In the smears and cultures made from the spinal fluid in each case the presence of diplococcus intracellularis meningitidis was proved.

Case XXIII.—Child 5 years of age, one of a family of six, the parents and four children who lived in four well-ventilated rooms of a tenement. The mother of the child was said to have been taken ill with a similar disease two days before the child was admitted to the hospital, and two days after his illness had begun. Nothing further was learned of her illness, as she was not brought to the hospital. The child died on the day after admission and five days after the illness began.

Case XXVII.—Boy 2 years and 8 months old. A sister 13 months old died on the day patient was admitted, after an illness of one week, with symptoms of vomiting, fever and convulsions. The family consisted of parents and five children, living in four rooms on the top floor of a tenement. The patient was perfectly well the evening before admission, and the attack began with sudden vomiting. The child remained in the hospital for seven weeks and finally died.

Case XXXI.—The patient's daughter, 7 years of age, was taken ill on March 18, five days after the onset of mother's illness, with sore throat and convulsions. The child was taken to the Presbyterian Hospital, and on April 10 was said to be convalescent, but deaf in both ears.

Case XLVI.—Physician, former member of House Staff. He had been run down and was worn out by his work when he was summoned to see a child with typical symptoms of cerebro-spinal meningitis, vomiting, rigidity of neck and convulsions. The child died in a few hours. His own symptoms began about one week after this possible exposure. This is the only instance in the series where the direct exposure of a person not living in the same family and surrounded by the same conditions of life was followed by the onset of the disease.

Here are eight instances in which the contagious nature of the malady may be considered. Do these cases indicate that the disease is contagious, or that the cases arising in the same family occurred as the result of exposure to the same source of infection?

All of the cases after admission to the hospital were treated in the open ward, and no attempt was made, in fact could not be made, to keep these patients separate from the others. No cases developed in the wards among patients suffering from other diseases, and none of the physicians or nurses in charge of the cases became infected.

All the cases occurring in children were treated in the First Medical Division and they were in no way separated from many other children in the ward, yet none of these children developed the disease. The only case which supports the theory of contagion as opposed to that of infection from the same source, is that of the former member of the House Staff above referred to. He lived, however, in a locality where a number of cases occurred, and although he was subjected to an undoubted exposure, it is also possible that he may have acquired the disease, as so many others did, from an unknown source.

The treatment in 33 of the 46 cases was general or expectant, and consisted in the use of ice-cap to the head, ice-bags to the spine, in some few cases baths, in nearly all the use of morphine, and where necessary the administration of stimulants.

In the 13 cases treated by the subdural injection of Lysol, these general methods of treatment were also employed in addition to the Lysol injections. In several cases the subcutaneous administration of an aseptic solution of Ergot was tried without, however, any apparent beneficial result. In one of the cases (XXIX.) the subdural injection of a 2 per cent. solution of Protargol was administered, with slight improvement following the first injection, but with no benefit as the result of the second injection, shortly after which the patient died.

A few words may be said giving more minute details of the 13 cases treated by the injection of Lysol into the sub-dural space. The solution used was the same as that employed in Lisbon, as reported in Dr. Seager's article in the *Lancet* of November 1, 1902, describing the epidemic which occurred in Lisbon in 1901-1902; namely, a 1 per cent. sterile solution of Lysol. The procedure was done with rigid antiseptic precautions. The needle, introduced for the purpose of obtaining the cerebrospinal fluid through the second or third lumbar space, was left in situ, and through this the solution of Lysol was injected, the amount varying according to the quantity of spinal fluid withdrawn, usually a few c. c. less of the Lysol being introduced than was withdrawn of the spinal fluid. In a few of the cases more Lysol was introduced than spinal fluid removed. The injection was made very slowly.

Of the 13 cases treated in this manner 8 died and 5 recovered; a rather better showing of recovery than in the cases treated with ice, morphine, etc. From so few cases, however, it is hardly warrantable to draw conclusions in regard to the efficacy of the Lysol treatment, especially as a number of equally severe cases not treated with Lysol made good recoveries.

A table indicating the cases treated with Lysol, the number of injections given to each case, the amount of spinal fluid withdrawn, the amount of Lysol solution injected, with the final result of the treatment, follows:

CASE NO.	DATE.	Amount Spinal Fluid Withdrawn.	Amount Lysol Solution Injected.	IMMEDIATE EFFECT OF THE INJECTION.	REMARKS.	Result.
VIII...	March 25	8 c. c.	6 c. c.	Temperature rose to 102.6	Death in seven hours	D.
XII...	" 28	15 c. c.	10 c. c.			D.
	" 30	40 c. c.	10 c. c.		20 convulsions	
XIII...	" 28	20 c. c.	8 c. c.	No bad effect	Complete recovery	C.
	" 30	10 c. c.	8 c. c.	Condition improved	Deaf in right ear	
	April 1	10 c. c.	8 c. c.			
XIV...	March 29	20 c. c.	6 c. c.	Restless during night	Condition grew steadily worse till death, April 1	D.
	" 31	7 c. c.	6 c. c.			
XV...	" 30	4 c. c.	5 c. c.	Condition improved		C.
	April 1	2½ c. c.	5 c. c.			
	" 3	7 c. c.	5 c. c.			
	" 5	3 c. c.	3 c. c.			
	" 9	7 c. c.	5 c. c.			
XVI...	March 30	10 c. c.	6 c. c	Condition better	Patient improved, but on May 26 she suddenly died	D.
	April 1	3 c. c.	5 c. c.			
	" 5	1 c. c.	1 c. c.			
	" 9	12 c. c.	10 c. c.			
XVII...	" 2	15 c. c.	10 c. c.	Patient restless	Condition grew worse till death	D.
	" 4	7 c. c.	5 c. c.			
XVIII...	" 4	3 c. c.	5 c. c.			C.
	" 6	15 c. c.	7 c. c.			
	" 9	25 c. c.	10 c. c.		Condition continued to improve. Cured	
	" 11	7 c. c.	5 c. c.			
XIX...	" 6	7 c. c.	5 c. c.			D.
	" 9	12 c. c.	10 c. c.	Improvement noted.	After April 25 patient grew worse till death.	
	" 11	30 c. c.	10 c. c.			
XXI...	" 9	7 c. c.	5 c. c.			D.
	" 11	18 c. c.	8 c. c.			
XXXI...	March 17	10 c. c.	10 c. c.		Patient made slow but continuous improvement	C.
	" 21	6 c. c.	6 c. c.			
	" 23	1 c. c.	None.			
XXXIX...	April 9	10 c. c.	10 c. c.	No apparent benefit		D.
XLIV...	March 7	10 c. c.	8 c. c.	Improved	Patient made slow but continuous improvement	C.
	" 23	30 c. c.	24 c. c.			

In 13 of the 34 fatal cases autopsies were performed (II., III., VIII., XI., XII., XVII., XIX., XX., XXII., XXIII., XXIV., XXXVI., XLIII.). In 11 of these cases the brain lesion was a purulent meningitis, involving both cortex and base. In the two cases where no pus was demonstrated the cortical vessels were much injected and there was a small amount of serum in the sulci (Cases XIX., XX.). In XIX. Lysol had been given and the patient had lived for about six weeks; in XX. death occurred twenty hours after the onset of the disease.

The cord was examined in four cases, II., XXII., XXIII., XLIII., and in all the meninges were found greatly congested and covered with a layer of pus.

In three cases (XI., XVII., XXII.) much purulent serum exuded from the foramen magnum on removing the brain from the skull.

In four cases in which autopsy was subsequently performed Lysol had been given; unfortunately, however, the cord was not examined in these cases and therefore it is impossible to state whether Lysol had any local action on the cord or its membranes.

Petechial ecchymoses of the serous membranes were noted in 5 cases (II., XI., XII., XX., XXXVI.). These ecchymoses were widely distributed, but were particularly noted on the pericardium and peritoneum.

The other pathological lesions noted were various, but the limits of this article do not permit of a more extended report of the findings at autopsy.

An analysis of the symptoms referable to the eyes and nervous system is interesting. In the 46 cases ptosis was noted only five times; strabismus was much more common and was reported in 24 cases; nystagmus was also very infrequent and was noted only four times. The condition of the pupils was recorded as "normal" in one case, "equal" in 15 cases, "irregular" or "unequal" in 11 cases, "contracted" in 5 cases, "dilated" in 10 cases. In 4 cases no notes on this point were made. The eye grounds were examined in only 5 cases, and in these were reported "normal" four times and "hazy" once. The presence of these eye symptoms does not seem to be an indication of the severity of the disease nor an aid in prognosis. Many of the cases in which recovery took place had well marked strabismus and irregularity of the pupils (Cases XIII., XV., XVIII., XXV. and XXXII.).

None of the patients who recovered were blind.

Headache was noted as the most marked initial symptom in 30 cases. In 16 cases it was not noted, and in these this was due to the fact that the patient was an infant (Cases III., IV., X., XX., XXVII., XXVIII.), or unable on account of mental condition to give information in regard to symptoms (Cases II., VIII., XVI., XXIV., XXXVI.).

The head was retracted in 21 cases and the neck was noted as rigid in 34.

Opisthotonos was seen but six times.

The superficial and deep reflexes were examined and notes made as follows: Superficial, "absent," 10 cases; "present," 6 cases; "increased," 8 cases; "diminished," 8 cases. Deep reflexes (patella), "absent," 12 cases; "present," 10 cases; "increased," 9 cases; "diminished," 10 cases.

Kernig's Sign was present in 29 cases, absent in 5 and not noted in 12 cases. Babinski's reflex was present in 5 cases, absent in 28 cases and not noted in 13 cases.

In 37 cases notes were made in regard to the presence or absence of the various forms of eruption noted in this disease. Herpes was present in 20 cases. In many of these the extent of the herpetic eruption was considerable. The most common location was the cheeks and lips; occasionally it occurred on the nose; once it was noted on the ear; once on the fingers and palm, and once on the wrist. In 2 cases the fluid from the vesicles was examined for the presence of the diplococcus meningitidis, but its presence was not demonstrated.

A petechial eruption was noted in 15 cases. In some of the cases it was profuse and in others only a very few petechiae were found. The size varied from that of a pin-head to a ten-cent piece. This eruption was present and profuse in all the foudroyant cases—those in which the disease was fatal in less than forty-eight hours (Cases II., VII., XI., XX.).

Blood examinations, chiefly leucocyte counts, were made in 37 of the 46 cases. In many of the cases a number of counts were made, the total number being 124. The lowest count made was in Case IV., where on one occasion, a few days before death, the leucocytes dropped to 2,500 per cmm. The highest count recorded was 64,800 per cmm., in Case V. In only six instances was the number of leucocytes below 10,000 per cmm. (Case IV., Case V., Case XVIII., Case XXXIV.). The average of the 124 counts was 24,570 leucocytes per cmm., far above the normal. In no single case was the average below 10,000, and the highest average count in a single case was 50,900.

In the cases which recovered the general tendency of the leucocyte counts was to return to the normal, though in several of the cases this gradual decrease in the counts was interrupted by some recrudescence or complication. For example: Case XIII. —Patient was allowed to sit up, with the result that temperature immediately rose, rigidity of the neck returned, there was vomiting and an increase in the leucocyte count. Case XVIII.—An erythematous eruption developed on ear and face, with the immediate result of an increased leucocyte count. Case XXVIII.—Patient suddenly developed a cough, and temperature rose to 104 degrees, but no physical signs in chest could be detected; the leucocyte count rose to 41,800; within a few days the count fell to 25,300, then to 21,200, when the child was discharged from the hospital at the mother's request.

The differential count of the leucocytes was made only twenty-seven times. The extreme activity of the service at the hospital during these months rendered it impossible for the interne staff to give the time necessary to make these careful records. As the result of these twenty-seven examinations the polymorphonuclear cells were found to be below 70 per cent. in eight instances; above 70 per cent. in nineteen. The highest polymorphonuclear percentage was 96 and the lowest 46. In other words, in two-thirds of the differential counts made there was a marked polymorphonuclear leucocytosis.

In the fatal cases the leucocyte counts ordinarily remained high, there being little tendency to a return to the normal.

The following table shows the range of the leucocyte counts:

Under 5,000	1
Between 5,000 and 10,000	5
Between 10,000 and 15,000	23
Between 15,000 and 20,000	25
Between 20,000 and 25,000	18
Between 25,000 and 30,000	18
Between 30,000 and 35,000	12

Between 35,000 and 40,000	6
Between 40,000 and 45,000	8
Between 45,000 and 50,000	3
Between 50,000 and 55,000	3
Between 55,000 and 60,000	1
Between 60,000 and 65,000	1

Ninety-six of the 124 counts therefore fell between 10,000 and 35,000.

The complete table of the leucocyte counts as made in the individual cases, with differential counts, follows:

CASE No.	DATE.	RED CELLS.	WHITE CELLS.	HAEMOGLOBIN.	DIFF. COUNT.
I..		....	No count.	....	
II..		....	No count.	....	
III..	February 13	....	22,600	....	
IV..	" 22	2,696,000	25,200	25 perct	Poly. 69 per ct. L. L. 30 per ct. S. L. 1 per ct.
	" 26	....	15,000	....	
	" 28	....	12,200	... .	
	March 2	....	24,100	....	
	" 7	....	28,200	....	
	" 14	....	24,800	....	
	" 21	....	13,900	....	
	" 29	....	2,500	....	
	April 1	....	9,200	....	Poly. 46 per ct. L. L. 37 per ct. S. L. 16 per ct. Myel. 1 per ct.
V..	March 5	....	64,800	....	
	" 9	....	45,000	....	
	" 14	....	42,000	....	
	" 21	....	31,000	....	
	" 29	....	5,400	....	
	April 1	....	8,800	....	Poly. 67 per ct. L. L. 30 per ct. S. L. 1 per ct. Eosin. 2 per ct.
	" 3	5,600,000	....	....	
VI..	March 5	....	22,000	... .	
	" 8	....	24,400	....	

Case No.	Date.		Red Cells.	White Cells.	Haemoglobin.	Diff. Count.
VI..	March	14	....	26,400	....	Poly. 91 per ct. L. L. 7.7 per ct. S. L. 0.6 per ct.
	"	21	....	20,800	....	
	"	28	....	17,800	... .	
	April	4	....	13,000	....	
	"	9	6,000,000	18,100	60 per ct	Poly. 80 per ct. L. L. 12 per ct. S. L. 8 per ct.
VII..	March	15	....	43,000	....	
VIII..	March	25	....	44,200	....	
IX..	March	24	....	16,500	....	Poly. 65 per ct. L. L. 31 per ct. S. L. 4 per ct.
	"	28	....	31,400	....	
X..	March	26	....	27,500	....	Poly. 80 per ct. L. L. 17 per ct S. L. 3 per ct.
	"	31	....	23,400	....	
	April	6	....	45,400	....	
	"	12	....	35,200	....	
	"	21	....	22,200	....	
XI..	March	26	....	26,200	....	
XII..	March	27	....	30,900	....	Poly. 85 per ct. L. L. 15 per ct.
	"	29	....	14,900	....	
XIII..	March	28	....	28,000	....	Poly. 85 per ct. L. L. 15 per ct.
	April	12	....	11,200	... .	
	"	15	....	23,600	....	
	"	18	....	18,600	....	
XIV..	March	29	....	50,900	....	Poly. 96 per ct. L. L. 4 per ct.
XV..	March	30	....	43,100	....	Poly. 90 per ct. L. L. 9 per ct. S. L. 1 per ct.
	April	9	....	14,600	....	
	"	12	....	15,800	....	
	"	21	....	18,000	....	
	May	12	....	16,000	70 per ct.	

CASE NO.	DATE.	RED CELLS.	WHITE CELLS.	HAEMOGLOBIN.	DIFF. COUNT.
XVI..	March 30	....	25,200	....	Poly. 46 per ct. L. L. 54 per ct.
	April 2	....	10,200	....	
	" 9	....	19,800	....	
	" 12	....	26,000	....	
	" 21	....	19,000	....	
	" 25	....	14,000	....	Poly. 62 per ct. L. L. 30 per ct. S. L. 8 per ct.
	May 12	....	11,000	55 per ct.	
XVII..	April 2	....	23,000	....	
	" 5	....	13,600	....	
XVIII..	April 5	....	28,000	....	Poly. 88 per ct. L. L. 12 per ct.
	" 9	....	15,600	....	
	" 15	....	34,400	....	
	" 18	....	19 800	....	
	May 12	....	9,200	70 per ct.	
XIX..	April 6	....	23.400	....	Poly. 80 per ct. L. L. 10 per ct. S. L. 10 per ct.
	" 12	....	13,000	....	
	" 20	....	22,000	....	
	,, 25	....	23,000	....	
	May 11	2,876,000	25,400	70 per ct.	
XX..	....	....	No count.	....	
XXI..	April 8	4,304,000	17,300	....	Poly. 60 per ct. L. L. 20 per ct. S. L. 20 per ct.
	" 12	....	35,000	....	
	" 15	....	12,600	....	
XXII..	April 10	....	33,000	....	Poly. 90 per ct. L.L. 7.5 per ct. S.L. 2.5 per ct.
	" 17	....	32,000	....	
XXIII..	April 12	....	14,000	....	Poly. 73 per ct. L. L. 12 per ct. S. L. 15 per ct.
XXIV..	April 14	....	40,300	....	Poly. 92 per ct. L. L. 5 per ct. S. L. 3 per ct.
	" 15	....	26,200	....	

Case No.	Date.		Red Cells.	White Cells.	Haemoglobin.	Diff. Count.
XXV..	April	17	4,888,000	19,300	75 per ct	Poly. 67 per ct. L. L. 22 per ct. S. L. 7 per ct. Eosin. 4 per ct.
	"	22	....	12,800	....	
XXVI..	April	19	....	57,000	....	Poly. 91 per ct. L.L. 7.5 per ct. S. L. 1.5 per ct.
	"	21	....	41,200	....	
XXVII..	April	22	....	27,200	....	Poly. 89 per ct. L. L. 8 per ct. S. L. 3 per ct.
	"	29	....	53,400	....	
	May	5	....	35,800	....	
	"	17	....	33,000	....	
XXVIII..	April	22	....	36,000	....	Poly. 87 per ct. L. L. 11 per ct. S. L. 2 per ct.
	"	29	....	41,800	....	
	May	5	....	25,300	....	
	"	12	....	21,200	....	
XXIX..	April	23	....	23,800	....	Poly. 87 per ct. L.L. 9.5 per ct. S. L. 3.5 per ct.
XXX..	March	14	....	40,200	....	Poly. 89 per ct. L.L. 2.5 per ct. S.L. 8.5 per ct.
	"	16	....	27,200	....	
XXXI..	March	22	....	37,800	....	
	"	23	....	32,600	....	
	"	24	....	26,200	....	
	"	26	....	22,400	....	
	"	28	....	38,600	....	
	"	31	....	12,000	....	
XXXII..	March	24	....	30,800	....	
	"	25	....	18,600	....	
	"	26	....	20,000	....	
	"	28	....	15,600	....	
	"	30	....	13,800	....	
XXXIII..	April	1	....	11,400	....	
XXXIV..	March	29	....	18,300	....	

Case No.	Date.	Red Cells.	White Cells.	Haemoglobin.	Diff. Count.
XXXIV..	March 30	....	18,100	....	
	April 1	....	12,500	....	
	“ 4	....	9,500	....	
XXXV..	April 15	....	10,000	....	
XXXVI..	April 28	....	19,800	....	
XXXVII..	April 27	....	50,800	....	
	“ 28	4,500,000	46,000	....	Poly. 94 per ct. L. L. 3 per ct. S. L. 3 per ct.
XXXVIII..		No counts made.	....	....	
XXXIX..		No counts made.	....	....	
XL..		No counts made.	....	....	
XLI..		No counts made.	....	....	
XLII..		No counts made.	....	....	
XLIII..	April 24	4,490,000	24,000	90 per ct.	
XLIV..	March 14	4,845,000	11,400	90 per ct.	
	“ 17	4,440,000	18,400	85 per ct.	
	“ 26	....	31,000	....	
	“ 28	4,120,000	33,000	85 per ct.	
	“ 30	....	27,400	....	
	April 1	....	18,600	....	
	“ 3	....	19,000	....	
	“ 7	....	17,200	....	
	“ 14	....	19,000	....	
	“ 24	....	14,200	....	
XLV..		No counts made.	....	....	
XLVI..	April 28	....	25,000	....	
	“ 29	....	33,600	....	
	May 6	....	26,600	....	Poly. 91 per ct. L. L. 5 per ct. S. L. 4 per ct.
	“ 13	....	16,200	....	
	June 4	....	12,200	....	
	“ 15	....	10,000	....	

The following account of the bacteriological examination of the spinal fluid has been written by Dr. Thomas Flournoy, who has been most interested in the cases and most careful in the investigation of them from the bacteriologist's standpoint. The writer wishes to thank Dr. Flournoy for his courtesy and valuable assistance in the preparation of this report.

Bacteriological Examination.

The fluid was withdrawn by lumbar puncture in the ordinary manner between the third and fourth or second and third lumbar vertebræ. It was received into sterile tubes and planted as soon as possible after drawing. The medium used in practically every case was a mixture, in plates, of agar and blood, in a proportion of about 8 to 1. From these plates the growth was transferred to Loeffler's blood serum, agar, broth, potato, etc. Where pneumococcus was found the growth was transferred to ascitic broth, and every strain of pneumococcus, including those isolated from the blood, was tested on mice, in order to demonstrate the presence of the capsule.

After withdrawal and transplantation the fluid was allowed to stand for a short time only, in order to obtain the coagulum, and from this smears were made and stained. In using Gram's stain the result was controlled by smears of colon or typhoid bacillus and staphylococcus or pneumococcus, placed upon the same slide with the smear from the fluid.

Case XXIV. was of bacteriological interest from the fact that, at autopsy, four days post mortem, pure cultures of the diplococcus intra-cellularis were obtained in three of five blood serum tubes, while crystals of ice were present on the meninges at the point where cultures were taken. These three cultures were all transplanted upon all of the ordinary culture media and definitely proven to be the organism stated.

Blood cultures were made five minutes after death in Case XI., and in three plates made by mixing 1 c. c. of the patient's blood with melted agar, twelve deep colonies developed. Five of these colonies were fished to the surface of the blood agar and again incubated. At the same time transplantations were made from each to agar and Loeffler's blood serum. In these cultures a small diplococcus was found, morphologically like diplococcus intra-cellularis meningitidis, decolorizing by

Gram's method and giving the characteristic growth of that organism on the three media mentioned. Unfortunately, no further tests were made, cultural or otherwise, so that it can only be stated as probable that it was the diplococcus in question.

Case No.	Date.	Amount Withdrawn.	Character of Fluid.	Smears.	Cultures.
II.	Feb. 21	Cultures made at autopsy.	Cloudy, white....	Intra-cellular, Gram-negative, biscuit-shaped diplococci in pus from spinal fluid.	Blood-agar plate cultures from fluid show diplococcus intracellularis meningitidis. Blood-serum tubes from surface of brain and from fluid in ventricle show same organism.
III.	" 24	......	....	Intra-cellular, Gram-negative, diplococci......	Pure culture diplococcus intracellularis meningitidis.
IV.	" 22	2 c.c.	Cloudy....	Pus-cells containing Gram-negative biscuit-shaped diplococci...........	Pure culture diplococcus intracellularis meningitidis.
V.	Mar. 5	½ c.c.	"	Pus-cells, intracellular diplococci...........	Pure culture diplococcus intracellularis meningitidis.
VI.	" 13	2½ c.c.	Very cloudy...	Pus-cells, intracellular, biscuit-shaped diplococci..........	Pure culture diplococcus intracellularis meningitidis.
	April 8	30 c.c.	....	....	Diplococcus intracellularis meningitidis.
VII.	Mar. 16	2 c.c.	Cloudy....	Pus-cells, intracellular diplococci..........	Diplococcus intracellularis meningitidis.
VIII.	" 24	1 c.c.	Milky.....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	" 25	Cultures made at autopsy.	....	....	Diplococcus intracellularis meningitidis from cerebro-spinal fluid.
IX.	" 24	2 c.c.	Cloudy....	Pus-cells, intracellular, oval, Gram—*positive*, diplococci........	Pure culture pneumococcus.
X.	" 27	2 c.c.	"	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XI.	" 26	3 c.c.	Bloody....	....	Diplococcus intracellularis meningitidis.
XII.	" 27	2 c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	" 30	40 c.c.	Turbid....	....	Diplococcus intracellularis meningitidis.
XIII.	" 27	½ c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.

CASE NO.	DATE.	AMOUNT WITHDRAWN.	CHARACTER OF FLUID.	SMEARS.	CULTURES.
XIII.	April 1	12 c.c.	Very slightly cloudy ...	....	Pure culture diplococcus intracellularis meningitidis.
XIV.	Mar. 29	20 c.c	Cloudy....	....	Diplococcus intracellularis meningitidis.
	" 31	6 c c.	Very cloudy, yellowish.	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XV.	" 30	4 c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	April 1	2½ c.c.	Slightly cloudy....	....	Diplococcus intracellularis meningitidis.
	" 3	5 c.c.	Cloudy....	....	Diplococcus intracellularis meningitidis.
	" 5	1 c.c.	"	....	Diplococcus intracellularis meningitidis.
XVI.	Mar. 30	8 c.c.	"	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	April 1	3 c.c.	"	....	Diplococcus intracellularis meningitidis.
	" 3	......	"	....	Diplococcus intracellularis meningitidis.
	" 5	......	"	....	Diplococcus intracellularis meningitidis.
XVII.	" 4	7 c.c.	Very cloudy, yellowish.	Pus-cells, intracellular diplococci	Diplococcus intracellularis meningitidis.
	" 6	Cultures made at autopsy.	....	....	From spinal fluid, diplococcus intracellularis meningitidis. From ethmoidal sinuses, staphylococcus pyogenes aureus.
XVIII.	" 4	3 c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	" 6	15 c.c.	"	....	Pure culture, same organism.
XIX.	" 6	7½ c.c.	"	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	" 9	12 c.c.	"	....	Diplococcus intracellularis meningitidis.
XX.	" 6	15 c.c.	"	Pus-cells, intracellular Gram—negative diplococci...........	No growth, plain agar plate planted 18 hours after withdrawing.
	" 7	Cultures made at autopsy.	....	....	Pure culture, diplococus intracellularis meningitidis from cerebrospinal fluid and from cerebral cortex.
XXI.	" 9	7 c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.

CASE NO.	DATE.	AMOUNT WITHDRAWN.	CHARACTER OF FLUID.	SMEARS.	CULTURES.
XXII.	April 10	1º c. c.	Very cloudy, yellow....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXIII.	“ 12	1 c.c.	Yellowish pus withdrawn by aspiration	Pus-cells, intra cellular diplococci..........	Diplococcus intracellularis meningitidis.
	“ 13	Cultures made at autopsy.	....	....	Diplococcus intracellularis meningitidis from spinal fluid.
XXIV.	“ 14	20 c. c.	Cloudy, yellow....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
	“ 20	Cultures made at autopsy four days post mortem.......	....	....	Pure culture diplococcus intracellularis meningitidis from cerebracortex and cerebrospinal fluid. Same organism with staphylol cocci from left tympanic antrum.
XXV.	“ 14	15 c. c.	Cloudy, white.....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXVI.	“ 19	½ c. c.	Cloudy....	Pus-cells, intracellular, oval, Gram—*positive*, diplococci......	Pheumococcus in pure culture.
	“ 20	......	Blood from vein......	....	Pneumococcus in pure culture.
XXVII.	“ 23	1 c.c.	Cloudy....	Pus-cells, intracellular diplococci...........	Diploeoccus intracellularis meningitidis.
XXVIII.	“ 23	7 c.c.	“	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXIX.	“ 25	3 c. c.	“	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXXIV.	Mar. 31	3 c.c.	Turbid....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXXVIII.	April 9	17 c.c.	Very cloudy....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XXXIX.	“ 10	14 c. c.	Very cloudy, yellow....	Pus-cells, intracellular diplococci...........	Diplococcus intracellularis meningitidis.
XL.	“ 19	1 c. c.	Slightly cloudy....	Pus-cells. No micro-organisms	No growth.
XLIII.	“ 25	15 c. c.	Slightly cloudy....	Few pus-cells. No micro-organisms	No growth.

The foregoing table may be summarized as follows:

Cultures from the spinal fluid obtained by lumbar puncture, or, in a few cases, at autopsy, were made and examined in 33 cases.

Pure pneumococcus was found in two of the cases, both of which were fatal (Cases IX. and XXVI.).

In two cases (XL. and XLIII.) no micro-organisms were found either in the smears or in the cultures.

In each of the remaining 29 cases the diplococcus intracellularis meningitidis was found in the culture, and in 28 of these cases was demonstrated in the smears made from the spinal fluid.

In concluding this report the writer desires to express his thanks to the House Staff of the First Medical Division for the interest and care with which they have followed the cases and for the time they have expended in making the various clinical examinations recorded.

THE BACTERIOLOGY OF THE BLOOD IN TYPHOID FEVER.

Being a Report on Sixty Cases and a Statistical Study.*

By Warren Coleman, M. D., and B. H. Buxton, M. D.,
OF NEW YORK CITY.
(*From the Loomis Laboratory of Cornell University.*)

Three years ago we undertook to study the bacteriology of the blood in cases of typhoid fever entering the wards of the Second Medical Division of Bellevue Hospital. Having records of 60 cases and having collected 544 others from the literature, giving a total of 604 cases, we feel justified in making a detailed study of the results that have been obtained.

We wish to express our thanks to Professors Thompson, Loomis and Dana for placing cases at our disposal.

The presence of typhoid bacilli in the blood of typhoid cases has been suspected for many years, but only recently has the search for them proved successful.

Burdach gives a summary of the blood examinations in typhoid fever, but has collected only the German and Italian cases, ignoring the American and French. The earlier attempts, 1885 to 1895, were mostly made with small quantities of blood taken from the finger tip and, as was to be expected, afforded almost uniformly negative results. Burdach mentions about 75 such cases, of which only 4 were positive. He himself studied 6 cases, taking only small quantities of blood, with negative results. The cultures from these early cases were frequently contaminated with skin cocci, and even the reported positive results must be looked upon with suspicion, since the tests for the typhoid bacillus were very imperfect, and until the introduction of the agglutination reaction no absolutely reliable one was known.

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*Read at a meeting of the New York Pathological Society, February 10, 1904.

All these earlier and more or less futile attempts have therefore been left out of consideration, and we have confined ourselves to the more recent cases, dating from 1895, when large quantities of blood have been taken from a vein and the bacilli obtained have been subjected to agglutination as well as to cultural tests.

Schottmueller takes 20 c. c. of blood, or as near that amount as can be obtained, and mixes each 2-3 c. c. with 6 c. c. of agar which is then poured into a Petri dish and allowed to set. He considers agar superior to broth, since in a solid medium the bactericidal action of the blood is less effective than in a fluid, and further, by counting the colonies, an estimate of the number of bacilli per cubic centimeter of the blood can be made.

Castellani first took blood from the finger tip and had negative results in all of 12 cases. Later he took several cubic centimeters of blood from a vein and distributed it among a number of flasks, each containing 300 c. c. of broth. After adopting this procedure, he was successful in 12 of 14 cases.

Busquet also sowed the blood in flasks containing 300 c. c. of broth, putting only a few drops into each. Dealing with such high dilutions he has been extraordinarily successful, as is pointed out elsewhere.

The American investigators have for the most part made comparatively high dilutions of the blood. We must admit, however, that in our own experiments we may have failed to prove the presence of bacilli in some instances by not sufficiently diluting the blood, and shall probably in future bring our methods more into line with those of Busquet.

Our practice has been to distribute 10 c. c. of blood into four flasks, each containing 100 c. c. of broth, and to incubate at 37 degrees C. In twenty-four hours there may be turbidity, though this does not usually occur until the second and may be delayed until the third day. As soon as a flask shows turbidity, a hanging drop is made from it and, if bacilli are found, a culture on agar is taken. From the latter further cultures are taken in glucose gelatine, litmus milk, neutral red agar and other differentiating media. The culture is also tested for its agglutinating properties in an active serum, and if it responds to all the tests is considered to be true typhoid. For agglutinating tests an immunized rabbit, which reacts up to 1:5000 or so with stock cultures, is always kept on hand, so that a sufficiency of serum for macroscopic observations can be obtained.

The rabbit is bled from the ear and 1 c. c. of clear serum mixed with salt solution in varying dilutions, usually 1:50, 1:200, 1:1000. One cubic centimeter of the dilution is pipetted into a small test tube and 1 c. c. of the culture in broth is added, making the test dilutions 1:100, 1:400, 1:2000. The tubes are put in the incubator and observations taken in one, two and three hours. If there is no reaction in three hours, the tests are considered negative. As soon as clumps visible to the naked eye are formed the reaction is marked +. When there is a distinct deposit at the bottom of the tube, but the fluid above is only partially clear, ++, and +++ as soon as the clumps have all subsided, leaving the fluid perfectly clear. When first isolated, the bacilli as a rule do not agglutinate readily and may not show the reaction at higher dilutions than 1:100. It has been found by experience, however, that a clear reaction at 1:100 is sufficient for diagnosis and may be relied upon.

From the bacteriological point of view, a few specially interesting points have been brought out in the course of this study:

1. We are able to confirm the observations of others, that when first isolated typhoid bacilli react less readily to immune serum than they they do late on, after having been grown on artificial media.

2. There are some, however, which are specially refractory, and this characteristic is often quite persistent, so that it may take several months of artificial culture before they will agglutinate readily.

3. The serum of rabbits immunized to such refractory strains quickly acquires agglutinating powers, but does not agglutinate its own strain any better than serum obtained by immunization with a strain which readily agglutinates. In other words, a good agglutinator will always agglutinate well, and a bad, badly, with any immune serum, no matter how it may have been obtained.

In our first cases, we used an apparatus devised by Dr. W. B. James for drawing the blood, consisting of a glass pipette, fitted at one end wth a needle and at the other with a piece of rubber tubing for making suction. But we abandoned this in favor of the Ermold antitoxin syringe, which we find much easier to handle and quite as easily sterilized.

To show at a glance the recorded cases, with their results, we give the following summary in tabular form. Believing, as we do, that paratyphoid infections should be considered clinically as cases of typhoid fever, we have included in our totals the cases of so-called paratyphoid fever in which bacilli have been found in the blood:

TABLE OF ALL CASES REPORTED.

NUMBER.	NUMBER OF CASES EXAMINED.	BACILLUS ISOLATED.	AUTHORS.
1	32	20	Ruediger.
2	14	1	Fox.
3	1	1	Warfield.
4	2	2	Stacey.
5	1	0	Pratt.
6	15	11	Cole.
7	25	21	Hewlett.
8	3	2	Johnston.
9	1	1	Gwyn.
10	2	2	Longcope.
11	2	1	Allen.
12	3	3	Hektoen.
13	1	1	Jochmann.
14	60	34	Buxton and Coleman.
15	25	17	Widal.
16	83	83	Busquet.
17	37	37	Courmont and Lesieur.
18	1	1	Brion and Kayser.
19	7	7	Troussaint.
20	119	98	Schottmueller.
21	6	4	Rodhain.
22	56	31	Kerr and Harris.
23	14	11	Castellani.
24	3	3	Koelzer.

NUMBER.	NUMBER OF CASES EXAMINED.	BACILLUS ISOLATED.	AUTHORS.
25	21	9	Kuehnau.
26	1	1	Thiemisch.
27	1	1	Tiessier.
28	2	2	Stern.
29	1	1	Sabrazes and Hugon.
30	1	1	Block.
31	2	2	Ferre and Antony.
32	2	2	Grandmaison et Cartier.
33	10	7	Auerbach and Unger.
34	2	2	Smith.
35	48	33	Warfield.
Totals	604	453	

The table shows the large proportion of 75 per cent. of 604 cases with bacilli in the blood at some stage of the disease, and we feel inclined to regard even this as somewhat lower than may be expected in future investigations, since the most recent records, those of Busquet and Courmant and Lesieur, show that with the most improved methods the bacilli are practically uniformly present in the first three weeks of the disease.

For sake of completeness from the bacteriological point of view, we give a separate table of cases in which paratyphoid bacilli have been obtained from the blood.

Table of Cases Showing Paratyphoid Bacilli in the Blood.

Number.	Day of Disease.	Day of Relapse.	Bacillus in Blood.	Author.
1	14	..	+	Ruediger.
	20	..	—	
	26	..	—	
	38	..	—	
2	13	..	+	
	20	..	—	
	27	..	—	
3	21?	..	—	Pratt.
4	11	..	—	Hewlett.
	..	4	+	
	..	10	—	
5	23	..	+	Johnston.
6	7	..	+	
7	10	..	—	
8	24	..	+	Gwyn.
9	10	..	+	Longcope.
10	15	..	+	
11	11	..	+	Allen.
12	14	..	—	
13	15	..	+	
14	5	..	+	Coleman and Buxton.
15	2	..	?	Brion and Kayser.
	7	..	+	
16	8	..	+	Schottmueller.
17	15	..	+	
18	5	..	+	
19	8	..	+	
20	21	..	+	

NUMBER.	DAY OF DISEASE.	DAY OF RELAPSE.	BACILLUS IN BLOOD.	AUTHOR.
21..............	14	..	+	Smith.
22..............	?	..	+	
23..............	?	..	+	

Some authors do not give the day of the disease on which the cultures were taken, so that the following study of the presence of the bacilli in the blood by week does not include all cases in the previous table. Further, many of the cases have been examined more than once, and in such instances each examination has been counted separately.

1. *Frequency in the first week.*

Eighty-five examinations have been made in the first week of the disease with positive results in 79, or 93 per cent. The earliest recorded positive result was obtained by Ruediger on the third day of the disease. Busquet and Troussaint report positive results on the fourth day, and a number of authors have found the bacillus on the fifth day of the disease.

2. *Frequency in the second week.*

One hundred and ninety-eight examinations have been made in the second week of the disease with positive results in 151, or 76 per cent.

3. *Frequency in the third week.*

One hundred and fifteen examinations have been made in the third week of the disease with positive results in 65, or 56.52 per cent.

4. *Frequency in the fourth week.*

Fifty-five examinations have been made in the fourth week of the disease with positive results in 18, or 32.72 per cent.

5. *Frequency after the fourth week, exclusive of relapses.*

Thirty-seven examinations have been made after the fourth week of the disease with positive results in 11, or 29.72 per cent.

The interesting fact brought out by this analysis is the large percentage of positive results obtained in the first week of typhoid fever, with the steady decline thereafter. It would appear from this that in typhoid fever the earliest and principal seat of infection is the blood, and that the disease may in truth be regarded as a septicemia. If this be true, we may in passing call attention to the utter futility of attempting to control typhoid fever by the administration of intestinal antiseptics. Of course this does not apply to the absorption of toxins other than typhoidal from and through the intestinal ulcers after these have formed.

We feel that the percentages in and after the fourth week, respectively 32.7 and 29.72, do not accurately represent the duration of life of the bacillus in the blood in the usual case of typhoid fever. In the first place, the cases are too few on which to base reliable conclusions, and in the second, the clinical course of the disease is unknown in practically all of them. Seventeen of our examinations were made on the 21st day or later, and in only three of them was the bacillus found. This, in a measure, accounts for the low percentage of postive results in our sixty cases. Courmont's experience agrees with ours in this regard. He believes that the bacilli disappear from the blood about the twentieth day. The convalescents we have examined did not show the presence of the bacillus in the blood. In the few cases where the clinical course is mentioned in the reports, the disease is marked as "severe," but the duration is not given. It is probable, however, that the examination of the blood was made in these cases for the very reason that the fever continued beyond the third week. Looked at from this point of view, the high percentages in and after the fourth week give an important clue to the unusual duration of the disease, and are as valuable in demonstrating the blood to be the principal seat of infection as the positive results in the earlier weeks.

The relation of relapses to the bacillemia is of such importance that a table of all the relapses which have been studied may be given. The interesting fact is brought out that in all but three of twenty-one relapses, or 14 per cent., the bacillus was found in the blood, indicating its reappearance. Here again, however, the number of cases is too small to attempt to draw reliable conclusions:

TABLE OF RELAPSES.

NUMBER.	DAY OF RELAPSE.	BACILLUS IN BLOOD.	AUTHOR.
1	3	+	Ruediger.
	16	—	
2	10	+	
3	39 of disease	+	
	45 "	—	
4	6	+	Warfield.
5	5	+	Hewlett.
6	3	+	
7	4	+	
8	4	+	Hewlett's paratyphoid.
	10	—	
9	8	+	Coleman and Buxton.
10	10	—	+ In original attack.
11	4	+	Rodhain.
12	?	+	Kerr and Harris.
13	?	+	Auerbach and Unger.
14	?	+	
15	?	+	Schottmueller.
16	3	—	Kuehnau.
17	?	—	Courmont and Lesieur.
18	?	+	
19	?	+	
20	8	+	Warfield.
21	10?	+	

Relation of Serum Reaction to the Bacillemia.

In 73 of the 604 cases, or 10 per cent., the bacillus was present in the blood before the serum reaction was obtained. This does not include the paratyphoid infections and must be regarded as lower than the actual percentage since in the majority of cases this reaction is not mentioned.

In the light of the knowledge which this study throws upon the subject, it would seem likely on *a priori* grounds that the bacillus is always present in the blood before the serum reaction develops. The typhoid bacillus produces soluble toxins in small quantity only; the toxins are liberated from the bodies of dead bacilli. Therefore, bacilli must invade the body and be in part destroyed before the anti-bodies and agglutinins can be formed. As is well known, the serum reaction does not appear till the end of the first or in the second week of the disease, yet, as has been pointed out, the blood is already swarming with bacilli. And in many instances the serum reaction does not develop till much later than the second week.

During the past two years we have had several applications for cultures of paratyphoid bacilli to test on cases of suspected typhoid with a negative Widal reaction. So far as we have heard the tests with the paratyphoid have been uniformly unsuccessful. In several of our own cases we have been inclined to suspect paratyphoid infection on account of the absence of Widal reaction, but bacteriological examination of the blood has shown the presence of true typhoid bacilli. In such instances the serum reaction to typhoid may not be positive before the twentieth to thirtieth days, and in one of our cases did not appear until the seventy-first day. We may also mention that Professor Thompson, in the fall of 1902, observing that several of his typhoid cases at the Presbyterian Hospital did not respond to the Widal reaction, asked one of us to make bacteriological examinations of the blood for him, as he suspected paratyphoid infection. Eight such cases were tested, two with negative results, whilst from six true typhoid bacilli were isolated. Professor Thompson has kindly permitted us to use these cases for the purposes of this article.

From these observations it would certainly appear that the absence of the Widal reaction, even to the third or fourth week of the disease, by no means implies that the illness is not typhoid or that the infection is due to paratyphoid bacilli. In such cases only a bacteriological examination of the blood can clear up the diagnosis.

The Diagnostic Value of Bacteriological Examinations of the Blood in Typhoid Fever.

In the average case of typhoid fever, neither the agglutination reaction nor a bacteriological examination of the blood is necessary to the diagnosis. Typhoid fever was recognized long before either of these tests became known, and it is probable that except in large centres or university towns that the great majority of cases are diagnosed, and correctly, to-day without these aids. The serum reaction may be obtained with dried blood or blood serum in a capillary tube sent to a distance, but a bacteriological examination of the blood demands not only complete laboratory facilities, but an experienced bacteriologist. Therefore we feel skeptical as to the general application of this aid to diagnosis. Yet the bacteriological diagnosis of typhoid fever is a distinct advance. It finds its applicability in just those cases where the clinical phenomena and even the serum reaction fail us, and, if future studies confirm the uniformly successful results of Busquet and Courmont and Lesieur, many doubtful cases may be cleared up. We think that little objection to the procedure need be apprehended on the part of the patients themselves or the patients' families, for in the majority of cases it is very simple and, by using ethyl chloride, may be accomplished practically without discomfort. However, as said above, it will be the exceptional case in which a bacteriological diagnosis will become necessary.

May the Colon Bacillus Cause a Primary Systemic Infection?

Since in two of our cases, Nos. 34 and 35, marked as negative in the tables, we found the bacillus coli communis in pure culture, the important question arises whether this organism is capable of causing a primary systemic infection, or whether its occasional presence in the blood is to be regarded as a secondary invasion. In the literature we have been able to find only one similar case. Fox, in 14 examinations of the blood, isolated the typhoid bacillus from one and the colon bacillus from another. Since there were several colonies of the latter in pure culture on his agar plates, it does not appear probable that their presence was due to accidental contamination. In this instance the serum of the patient became positive to Widal on the thirty-second day, though it had previously been negative, so that there may have been a mixed infection.

Under the title of Colon Bacillus Infection, Burch reported, in 1902, a series of cases presenting typhoidal symptoms which lasted from 7 to 10 days. The Widal reaction was negative (dilution not stated) in every case throughout its course. In many of the cases the urine was turbid and swarmed with motile bacilli which responded to the cultural tests for B. Coli communis. The sera of the paitents uniformly gave a complete reaction with the bacilli obtained from the urine, but here again the important fact of the degree of dilution is not stated.

Evans and Sailer, of Philadelphia, likewise mention a case of apparent typhoid, where the serum in the third week was positive at 1:150 with a culture of coli commuis obtained from Paris, but only gave an imperfect reaction with typhoid at 1:10. No blood cultures were taken, but it seems likely that this was an instance of colon infection.

In case 34 of our series the colon bacillus was found in the blood on the eighth day of the disease, the serum reaction against typhoid having been negative on the sixth day. As, however, the reaction became positive on the ninth day, the presence of the colon bacillus must be regarded as part of a mixed or secondary infection. The case ran a very mild course.

In case 35 the colon bacillus was found on the tenth day and the serum reaction against typhoid was negative as late as the twlefth. This case also was exceedingly mild and was of short duration. Unfortunately, owing to vacations, the bacillus was not identified as coli communis until the case had left the hospital, so that its bearing on the question of systemic colon infection, while suggestive, was not determined.

In order to make our own cases available for future statistical studies, we append the following table, giving the more essential facts concerning them:

NUMBER.	DAY OF DISEASE.	DAY OF RELAPSE.	BACILLUS IN BLOOD.	SERUM REACTION.	REMARKS.
1..........	21	..	—	+	
2..........	40	..	—	+	Convalescent.
3..........	..	8	+	..	Relapse.
4..........	14	..	+	—	
5..........	21	..	—	—	
6..........	60	..	—	+	Convalescent.
7..........	5	..	+	—	Paratyphoid.
	27	..	—	..	
8..........	14	..	+	..	
9..........	28	..	—	+	
10..........	21	..	—	—	Diagnosis doubtful.
11	14	..	—	+	
	19	..	—	..	
12..........	8	..	+	—	
	20	..	+	..	Serum reaction + with own bacillus, 1:80.
13..........	8	..	—	—	Blood clotted in tube.
	13	..	—	..	
	19	..	—	..	
	27	..	+	+	
14.........	14	..	+	+	
15..........	14	..	—	+	
16..........	8	..	+	+	
17..........	14	..	+	+	
18..........	14	..	+	+	
19..........	8	..	—	..	
	13	..	+	..	Agglutinates badly.
20	11	..	—	—	
21..........	11	..	—	+	
22..........	8	..	+	+	Agglutinates badly.
23..........	21	..	+	+	

NUMBER.	DAY OF DISEASE.	DAY OF RELAPSE.	BACILLUS IN BLOOD.	SERUM REACTION.	REMARKS.
24	14	. .	—	. .	
	21	. .	—	. .	
25	14	. .	—	—	
26	14	. .	+	—	
		10	—	. .	Relapse.
27	14	. .	+	—	
28	8	. .	+	—	
29	. .	. .	—	. .	
30	. .	. .	—	+	
31	. .	. .	—	. .	
32	11	. .	+	+	
33	9	. .	+	+	
34	8	. .	—	. .	Colon bacillus.
35	10	. .	—	. .	" "
36	8	. .	+	+	
37	8	. .	—	—	
	25	. .	—	. .	Serum reaction + 35th day.
38	12	. .	+	+	
39	38	. .	—	—	Serum reaction + 71st day (Dr. Dow's case).
40	14	. .	+	—	
41	13	. .	+	+	
42	12	. .	—	+	
	14	. .	+	+	
43	10	. .	—	+	
44	8	. .	+	—	
45	7	. .	+	—	Dr. Nammack's case.
46	26	. .	—	—	" " "
47	10	. .	+	+	
48	10	. .	+	—	
49	8	. .	+	—	

NUMBER.	DAY OF DISEASE.	DAY OF RELAPSE.	BACILLUS IN BLOOD.	SERUM REACTION.	REMARKS.
50	23	..	—	+	
51	11	..	—	—	
52	19	..	+	—	Dr. Thompson's Presbyterian Hospital case.
53	29	..	—	—	Dr. Thompson's Presbyterian Hospital case.
54	7	..	+	—	Dr. Thompson's Presbyterian Hospital case.
55	21	..	—	—	Dr. Thompson's Presbyterian Hospital case.
56	9	..	+	—	Dr. Thompson's Presbyterian Hospital case.
57	21	..	+	—	Dr. Thompson's Presbyterian Hospital case.
58	16	..	+	—	Dr. Thompson's Presbyterian Hospital case.
59	11	..	+	—	Dr. Thompson's Presbyterian Hospital case.
60	10	..	—	..	

In conclusion, we wish to express our indebtedness to Dr. Letchworth Smith for his kindness in isolating the bacilli in a number of cases.

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SOME STATISTICS AND STUDIES FROM THE ALCOHOLIC WARDS OF BELLEVUE HOSPITAL.

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The following statistics of the alcoholic wards in Bellevue and the studies accompanying them have been compiled in the hope that they may be of service to those who wish to study the subject of alcoholism as it is presented in a large hospital service in a cosmopolitan city to-day. Few realize the extent of the alcoholic service in Bellevue Hospital, and that one-quarter of the total admissions to the hospital pass through the alcoholic wards. The statistics here offered are based on some of the data of the nine years from 1895 to 1903, inclusive. It has been impossible to obtain full records in all details for the nine years, but even the partial data obtainable are so large that they seem of value for study. My personal interest in the study of alcoholism has been only that of a general practitioner of medicine and not that of a specialist in nervous diseases nor of an alienist. The clinical picture of disease in Bellevue Hospital is so complicated by the morbid processes produced by alcohol that it seemed necessary to study carefully the alcoholic patients in order to clearly appreciate the medical service in the general medical wards of the hospital. The statistics here presented may interest a specialist, the studies are on the pathology and treatment of delirium tremens and allied conditions. The following table shows the total admissions to Bellevue Hospital for 1895 to 1903, inclusive, and the proportion of admissions to the alcoholic wards:

YEAR.	TOTAL ADMISSIONS.	TOTAL ALCOHOLICS.	PER CENT. OF ALCOHOLIC ADMISSIONS.
1895	16,392	4,190	25.5
1896	19,967	5,038	25.2
1897	20,590	5,714	27.7
1898	22,866	6,151	26.9
1899	22,267	5,752	25.8
1900	24,315	5,133	21.1
1901	23,420	5,454	23.4
1902	23,073	5,830	25.2
1903	27,068	8,370	30.9
Totals	199,958	51,632	25.7

The curve of admissions for the alcoholics does not run parallel to the curve of the general admissions. This is shown comparatively in Chart I. This is made from the daily average admissions to the alcoholic wards compared with the daily average admissions of males and females over 12 years of age in the general wards of the hospital. The figures were obtained by taking the total admissions to the hospital for each month of adults over 12 years of age and subtracting the alcoholic admissions for each month, and calculating the daily average for the nine years. The curves are nearly reversed, the general admission curve being high when the alcoholic curve is low and falling as the alcoholic curve rises. The two female curves agree in an April and October rise and a November fall, and the two male curves agree in a May and October fall and a November rise.

Considering the curve of the alcoholics by months we have the monthly admissions for the nine years in Table II., and also the monthly average and the daily average. The accompanying chart gives the curves for each year and the average curves for the nine years for both sexes. It is noticeable that the point of the January curve is usually lower than that of the preceding December. There is always a fall in February, often more apparent than real, because of the shortness of the month; but still, on the average, the fall is real, as seen in the daily averages. There is usually an early spring rise, a May fall and a June rise and a midsummer fall, and then a rise reaching the highest daily average in November in the male and October in the female curve. The sudden great rises in August, 1896, and June, 1897, are due to a sudden and intense heat which occurred at those times in New York. At the end of 1902 and during 1903 the large increase in numbers was due to the idleness and despondency caused by no work among the building trades, owing to a prolonged strike. I have no explanation to offer for the variations in the female curve in 1899. The curves here shown do not agree with those given by Bonhoeffer, who has found in Breslau, and quotes Näcke as having found essentially the same in Königsberg, that January, February and March show the lowest figures, and a steady rise from April, reaching the highest figures in July and August, remaining about the same in September, and falling off sharply in November and December. Bonhoeffer gives as the explanation for this that the burning up of the alcohol takes place slower in hot weather than in cold, corresponding to the physiologic experience that the exchange of gases from the lungs is in inverse ratio to the external temperature. He believes also that the slower oxydation of alcohol in the organism during warm weather is the reason for the greater number of delirious patients in later summer months. This may be one factor to account for the September rise in the Bellevue curves, but it does not explain the maximum rise in October in the female and November in the male. In my experience the winter service has shown more severe cases of delirium than the summer service, as well as a greater number, due, it has seemed to me, to the greater consumption of alcohol for bodily fuel as the cold nights came on, hence a severer poisoning from the greater amount imbibed. The variations in the Bellevue curves show more varied and wider excursions, as a rule, in the male than in the female curves, and the rises and falls of the two curves in the same years do not always run parallel. There are evidently sociologic factors to be considered as well as meteorologic, and apparently the same forces do not act simultaneously on the two sexes. It would also seem that whatever the causative factors are which produce the variations in the curves, they act with more even force on the women than on the men. It is also probable that the causative factors vary for the two sexes. Taking the curves of the daily averages by months for the nine years, it is striking how the two curves coincide until October is reached; then they are reversed until from December to January the two again show a coincident fall. The reason is not clear for the sudden divergence of the two curves. Although divergent, the two curves agree in showing the greatest number of admissions for the year in the last four months. This agreement is probably due to the effect of the beginning cool weather after the summer heat and the greater variability of the weather during these months, causing a greater consumption of alcohol. This would account for increase in numbers in the last three months, but not for the sudden rise in September. Sudden heat causes an increase in the alcoholic admissions, and this probably is one cause for both the June and September elevations in the two curves.

Studying the admissions from the standpoint of nationality and comparing them with the same nationalities as admitted in the general wards during the same periods we have the following tables:

	MALES.				FEMALES.			
	General Admissions.	Alcoholic Admissions.	Per Cent. of General Admissions.	Per Cent. of Alcoholic Admissions.	General Admissions.	Alcoholic Admissions.	Per Cent. of General Admissions.	Per Cent. of Alcoholic Admissions.
United States.............	13,716	5,567	44.41	52.34	16,556	3,231	42.34	38.48
Canada....................	204	138	0.66	1.29	328	87	0.83	1.06
Ireland....................	5,289	3,273	17.12	30.77	9,583	3,730	24.45	46.11
Germany..................	2,849	717	9.23	6.74	3,261	221	8.32	2.73
England..................	785	423	2.54	3.98	1,261	419	3.21	5.18
Scotland..................	273	152	0.88	1.43	370	160	0.94	1.98
France....................	198	43	0.64	0.11	.....	.....	.....	....
Italy......................	1,533	43	4.96	0.44	.....	.....	.....	....
Other nationalities........	6,035	280	19.54	2.63	7,831	328	19.98	4.05
Totals..............	30,882	10,636	99.98	99.98	39,190	8,089	100.00	99.99

Considering the above tables in detail it is noticeable that in the general admissions the foreign born predominate over the native born in both men and women, but in the alcoholic admissions this is true of the women and the reverse is true of the men. Taking the two sexes together, we have in general admissions, foreign born 57 per cent., native born 43 per cent.; in alcoholic admissions, foreign born 53 per cent., and native born 47 per cent. From the entries at my disposal it has been impossible to separate in the native born those of native parents from those of mixed or foreign parentage, and, as a matter of fact, among the men about 20 per cent. of the native born have Irish names. Comparing these percentages with those of the whole of New York City, we have from the United States Census for 1900, in the 3,437,202 inhabitants, 63 per cent. native born and 37 per cent. foreign born. In the Borough of Manhattan there were in 1900 59 per cent. native born to 41 per cent. foreign born, showing that the foreign born are disproportionately represented in the hospital admissions, both in the general and alcoholic admissions. It is also noticeable that the ratio of Irish, Canadians, English and Scotch is greater among the alcoholic than in the general admissions, while the reverse is true of the Germans and other nationalities. In the alcoholics, under the heading of other nationalities, there are some twenty-six different nationalities represented, but the number of individuals in each did not seem sufficiently large to draw any conclusions from them. The figures in the table for males under France shows this, though the figures under Italy are more significant. The Scandinavian races were not classified in such manner that I could obtain accurate figures for the general admissions. Taking the foreign born alone and calculating the percentages of the nationalities here given, it is strikingly noticeable that the total number of Irish form 71 per cent. of the alcoholic admissions among the foreign born. Among the general admissions they form 38 per cent. of the other nationalities. The Canadians form 2 per cent. of the alcoholics to 1 per cent. of the general admissions; the English 8 per cent. to 5 per cent.; the Scotch 3 per cent. to 2 per cent.; the Germans 9 per cent. to 15 per cent.; other nationalities 7 per cent. alcoholics to 39 per cent. general admissions. Of the nationalities here in the general admissions Italians and Russians form a large number. Comparing these percentages to the percentages of these nationalities among the foreign born in New York City, the United States census for 1900 gives of the total 1,270,080 foreign born in New York City Canadians, 1.7 per cent.; Irish, 21.7 per cent.; English, 5.4 per cent.; Germans, 25.4 per cent.; Scotch, 1.6 per cent.; French, 1.2 per cent.; Italians, 11.5 per cent.; Russians, 12.2 per cent. Archdale Reid states that in England he has never seen a Hebrew with delirium tremens. In 11,854 entries among the male alcoholics there were 99 Hebrews, and among 7,928 female alcoholics there were 72 Hebrews. It is very noticeable how few negroes are admitted in the alcoholic wards. Among 15,285 male alcoholics there were 11 negroes, and among 7,928 female alcoholics there were 9 negresses. Among the male negroes I personally can remember but 2 who had true delirium tremens. To sum up, it appears that among the male alcoholics the nationalities occur in the following order: Native born with about 20 per cent. Irish names, Irish, German, English and Welsh, Scotch, Canadian, Scandinavian and Russian. Among the female alcoholics the order is Irish, native born, English and Welsh, German, Scotch, Canadian, French, Russian and Scandinavian.

The civil condition in 8,930 men was given as single, 5,424; married, 2,578; widowed, 928. In 7,776 women it was given as single, 1,740; married, 3,526; widowed, 2,510. These figures for the women are not as accurate as for the men, as many prostitutes declare they are married or widowed when such is not the case. What this percentage of inaccuracy is it is impossible to state. It may be well to state here, as to the probable accuracy of the statistics here given: Of the admissions in the male alcoholic ward about 1.7 per cent. are drug habitués and other non-alcoholic conditions, and in the female ward about 4.9 are non-alcoholics. Besides the alcoholics and drug habitués there are epileptics and insane, but of the individuals recorded here as alcoholics there is no doubt as to the diagnosis. All drug habitués, epileptics, insane and even alcoholics who were taking morphine or cocaine have been carefully eliminated.

The number of repeaters who are continually readmitted was 12 per cent. about six years ago, but of late years and at present is much less. Some years ago I remember one man who was admitted forty times in nine months for varying degrees of alcoholism. While the repeating has diminished the actual number of admissions has increased, so that the percentage of repeaters in the statistics here given is small.

In considering the ages of the alcoholics, Chart III. shows the curves for the two sexes in quinquennial periods. All age statistics show a tendency to concentrate on the ages ending in 0 and 5; hence, by making the figures ending in these numbers the centres of each period, a more even and accurate estimate is obtained. Chart III. shows the ages in percentages of each period to the total number, so that the curves are on the same scale. It is noticeable that the greatest number of women are between 28 and 32, and the greatest number of men occurs five years later. 37.9 per cent. of the women and 27.5 per cent. of the men are younger than 33 years, and 54.5 per cent. of the women and 47.5 per cent. of the men are younger than 38. In the old age periods, after 57 years of age, there are 7.6 per cent. of the women and 6.4 per cent. of the men. From 33 through to 57 years of age there are 66 per cent. of the men and 53.8 per cent. of the women. In youth and old age there is a greater relative percentage of women than men; in middle life there is a greater relative percentage of men than women. Considering age in relation to nationality it is noticeable that there are twice the number of women below 28 years of age among the native born than among the Irish, but the Irish greatly predominate in the later years of life. It is unfortunate that the time at my disposal has not permitted me to work out the relation of age to nationality among the men. It was very noticeable, however, in working out the relation of age and nationality to occupation among the men that the Irish also predominated in the later years of life.

Considering next the occupations of the alcoholic admissions, we have but relatively few women to judge from, there being but 2,286 entries in which the occupation is given with age and nationality. Either one or the other is deficient in some 443 others in which the occupation is given, making a total of 2,729. Considered from the occupation alone, we have housework, 924; domestic, 595. In these two classes are comprised the many prostitutes that come and hide their real vocation under these occupations. Of the domestic servants where definite service is mentioned, there were laundresses, 174; cooks, 131; housekeepers, 51; waitresses, 38; chambermaids, 13; ladies' maids, 9. Of the dressmakers there were 100, seamstresses 75, milliners 16. Of the cigarmakers there were 10, of the others under this heading there were 21 different occupations, with 1 to 4 individuals under each heading. Of the miscellaneous occupations there were 16 occupations. Of these 20 were actresses, 14 telephone or telegraph operators, 11 trained nurses, 6 teachers (3 music teachers, 3 school teachers), 4 journalists, 4 hairdressers, the other occupations having 1 or two representatives. Housewife means married women living with their husbands or widowed mothers living with their sons. The number of individuals in specified occupations here given is too small to draw any general deductions beyond stating that it is impossible to say how many prostitutes comprise the housework and domestic class, but there is no doubt that they comprise a large number of the younger women, the older women being the real domestic servants. Of these servants it is evident that laundresses and cooks are more prone to alcoholism than the other forms of service. Dressmakers and seamstresses are more often alcoholic than women working in factories or than those working in shops and stores. Of the 16 miscellaneous occupations actresses, telegraph and telephone operators, and trained nurses comprise more than half of the total in this group.

The two tables of the occupations of the male alcoholics, one according to nationality, the other according to ages, are in the main self-explanatory. The column at the right of the age group is the ratio per 1,000 between the admissions here given for each occupation and the total number of males in each occupation, as given for the boroughs of Manhattan and The Bronx, of New York City, in the United States census for 1900. This, of course, gives only an approximate accuracy, but it seems the best method for a basis of comparison between the different occupations as to the relative occurrence of alcoholism in these various occupations, especially as these two boroughs are the territory from which the vast majority of the patients come who are admitted into Bellevue Hospital, and also the admissions here studied occurred from December 12, 1899, through 1900 and January, 1901, and from August 1, 1902, to October 21, 1903. The 1900 census has been followed for tabulation of the occupations.

In the professional class the great difference of the ratio of journalists and actors compared with the others of this class is very striking. The irregularity, strain and mental excitement of their existence probably accounts for this. In the commercial and mercantile class it is noticeable that while bookkeepers, clerks, etc., are present in large numbers, their ratio is not excessive and their age curve shows a maximum younger than the general age curve. This age curve is very pronounced in the stenographers, and their ratio is high. In recording commercial travelers and salesmen only 9 were entered as commercial travelers, the ratio here given is for the 367 salesmen. The merchant and dealer class is for wholesale and retail and small dealers. These entries are not all due to inhabitants of New York City. This is one of the classes in which there are many Southerners and men from other States who, coming to New York on business, go on a spree and break it off in Bellevue. In the bartender, liquor dealer and saloon keeper class the ratio, though high, would be very much higher for bartenders and lower for the other two if these three occupations had been kept separate. The census groups waiters and servants together, and the ratio here given is for both combined. The laborers here are the general unskilled workers, including packers, porters, etc. In the class of manufacturing and mechanical trades it is noticeable that the ratios for the manufacturing range distinctly lower than for the trades. In the occupations of transportation and outdoor work it is very noticeable that the ratios are high, with the exception of the steam and street railroad employees, in whom it is noticeably low. Boatmen and canal men, sailors, fishermen and oystermen I have had to group as one class to obtain their ratio.

This ends the purely sociologic statistics. Let us consider these alcoholic admissions from a purely medical standpoint.

Since Anstie, forty or fifty years ago, classified alcoholism as a nervous disease it has been so considered and treated. A close study of the action of alcohol in the body and the lesions it produces will, I think, cause us to realize that the symptoms referable to the brain are not the ones demanding urgent treatment, and are secondary in importance when the acute and chronic poisoning is under consideration. As far as the relationship of a man to his environment is concerned, the effect of alcohol on the brain is the predominant manifestation; but as far as the continuance of the individual's existence is concerned, the effect on the heart and circulation is the most important. All the viscera are affected in the lesions of chronic alcoholism, the cerebral symptoms dominating the clinical picture only because of the special functions of the brain.

Before considering the clinical symptoms and types of alcoholism, let us consider the action of alcohol on the body and the lesions it produces. Aside from the local action on the mucous membranes, the first action of alcohol is that of causing a flushing of the face by dilating the capillaries. The cause of this primary dilatation is unknown and various hypotheses have been advanced in explanation. It may be a beginning paralysis of the vaso-constrictors too delicate to be measured by our present instruments; or it may be a stimulation of the vaso-dilators; or a stimulation of some inhibitory function acting on the vaso-motor centre and inhibiting the tonus of this centre. Moderate doses cause a change in distribution of the blood by further dilating the peripheral capillaries without causing any change in blood pressure and without any stimulation or depression of the heart's action. Large toxic doses greatly lower the reflex excitability of the vaso-constrictor centres, dilate the arteries and capillaries of the splanchnic and peripheral areas, causing a lowering of blood pressure, and also act directly on the heart muscle as a powerful depressant, weakening first the auricular and then the ventricular systole, causing more or less distension of both cavities, marked slowing of the movements and great diminution in the output of blood. This action on the heart becomes of great importance when the cardiac muscle is much impaired by fatty degeneration and is further poisoned by alcohol, and is doubtless the cause of the not infrequent sudden death seen in certain young alcoholics, especially young women.

The dilatation of the skin capillaries gives a sense of warmth after the ingestion of alcohol and there is an increased radiation of heat from the body. The temperature of the body is seldom reduced more than ½ degree or 1 degree C., though very large quantities of alcohol may cause a fall of 3 to 5 degrees C., or even more, as in one of my patients, whose temperature on admission was 90 degrees F., and remained between 90 degrees F. and 92 degrees F. for fifteen hours, then rose slowly to normal, reaching this point thirty-six hours after admission. The lowest temperature I have seen was in a woman who was found in a hallway comatose from alcohol; the temperature of the air was about 6 degrees above zero. After the patient had been in the hospital for three hours wrapped in blankets and surrounded with hot water bottles, her temperature in the rectum was 80 degrees F. This temperature I took by inserting a thermometer and keeping it in the rectum for ten minutes. The temperature four hours later was 83 degrees F. The temperature reached normal twelve hours after admission and continued to rise, until twenty-four hours after I had first taken it it was 104 degrees F. The patient died thirty-six hours after admission with a temperature of 103.8 degrees F. Post mortem examination showed two small patches of broncho-pneumonia in left upper and right lower lobes. Such a temperature as this in drunkards exposed to cold is unusual, but by no means unique. Schafer's Physiology quotes records of temperature in drunkards, after such exposure, of 79.5 degrees, 80.6 degrees, 83.1 degrees F., the patients dying; others with temperatures of 75.2 degrees, 76.4 degrees, 82.2 degrees and 86 degrees F., and the patients recovered after a few days.

The respiration is stimulated directly, according to Binz, by alcohol in moderate doses, more so in fasting and fatigued individuals than after a meal or in non-fatigued persons. This direct stimulant action on the respiratory centre is not accepted by the majority of experimenters, and alcohol is usually considered an indirect stimulant. In large toxic doses there is a paralyzing effect on the respiratory centres and the breathing becomes stertorous and slowed.

From the experiments of Chittenden and his assistants we know that alcoholic fluids increase the flow of an active saliva while the fluid is held in the mouth. Even 5 per cent. of absolute alcohol mixed with saliva slightly increases the amylolytic action; large amounts retard this action, but even 10 per cent. produces only slight retardation in the amount of sugar formed. Whiskeys, wines and malt liquors, however, strongly inhibit the salivary digestion because of their acid properties. Gastric secretion is increased by a single dose of alcohol, and even after the alcohol is absorbed from the stomach or intestines it increases the secretion of an active, acid gastric juice. The absorption of the alcohol by the stomach occurs rapidly. On the proteolytic action of the gastric juice until 5 to 10 per cent. of absolute alcohol is present there is no retarding action, but 15 to 18 per cent. reduce the digestive action to one-fourth or one-third. Whiskey, brandy, gin and rum act in ratio to their alcoholic content. Sherry and clarets inhibit proteolysis out of all proportion to their alcoholic content, due to solid matters in the wines. The Rhine wines, for the same reason, retard when present in 10 per cent. Malt liquors in small amounts rather increase than retard the rate of digestion; but large amounts inhibit proteolysis, not by their alcoholic content, but by the comparatively large amounts of extractives they contain. Pancreatic digestion is more sensitive to alcohol and to the acid bodies contained in alcoholic beverages than is gastric digestion. Still it is doubtful if much alcohol reaches the intestinal tract, it being too rapidly absorbed. Moderate doses of alcoholic beverages, taken not too frequently, seem as a sum total of their action to favor an increase in the digestive processes; but after repeated consumption the digestive processes are blunted and a diminution in digestion occurs. After the atrophic gastritis has begun there is a diminution or even absence of secretion of

HCl. Moderate doses seem to slightly favor intestinal absorption and toxic doses to disturb this function.

After the absorption of alcohol we know that most of it—98 per cent. of moderate doses—is consumed in the body, the residue being excreted by the lungs and kidneys. It is used economically by the body for the needs of heat and muscular work, following the laws of the conservation of energy. It is used like its isodynamic quantities of carbohydrates and fats, and in this way always protects the body fats from consumption. It can and sometimes does protect the protein of the food and body from consumption. At times, in small doses, especially in persons unaccustomed to its use, it fails to protect the proteid, even increasing the protein disintegration. In large quantities it is positively toxic and may retard or even prevent metabolism and proteid metabolism in particular. This action on metabolism shows clearly the double action of alcohol as food and drug. Unfortunately, while the food action may predominate when small doses are ingested, the drug and toxic actions predominate with large doses.

The action of alcohol on the brain is still a subject of dispute. Binz holds that alcohol first stimulates and then depresses; Schmiedeberg, Bunge and others, that the apparent stimulation is really a paralysis of the higher functions and that alcohol depresses from the beginning. Kraepelin holds that, while all reactions requiring nicety of judgment are dulled by even small doses, in the early stages of its action alcohol truly stimulates the motor functions. This is also denied. Alcohol in moderate doses does not increase the quantity or vigor of mental processes. It clearly tends to lessen the power of clear and consecutive reasoning and decidedly lowers the acuteness of the several senses. After larger doses the judgment is lost; the power of self-control and will are in abeyance; all sense of proportion is gone; the sense of responsibility and restraining impulses is destroyed; and finally the motor power for speech and motion disappear and torpor and coma supervene. The result of continued action of large doses is a permanent loss of these mental functions, and the chronic alcoholic becomes an irresponsible animal, stirred only to act for selfish gratification, not recurrently drunk because he drinks, but ever drinking that he may remain drunk and oblivious to his environment.

This, then, is the action of alcohol on the living organism; the results of this action are next to be considered. To study the lesions resulting from the excessive use of alcohol I have taken the post mortem reports of 125 hospital histories. These histories were chosen not because they showed any given lesions, but because the patients gave a definite history, in varying degrees, of alcoholic excesses, 90 being those of men, 35 of women. Many histories with alcoholic pneumonia have purposely been rejected. With the kind assistance of Dr. Harlow Brooks, late Curator of the Fourth Division of Bellevue, the lesions found in each viscus have been tabulated, and the facts thus found will aid greatly in understanding the clinical symptoms and in determining the choice of methods of treatment.

The following table of the age at death of 541 alcoholic patients shows some interesting differences in the two sexes. The men seem to live longer than the women in this table, but that is in contradiction to the general age curves given above. This may be explained by the fact that here the record is of deaths caused by the alcoholism or pneumonia developing in the chronic alcoholic, while in the general age curve the record is of admission of acute and chronic alcoholism or an acute exacerbation of a chronic condition, which conditions are not necessarily fatal.

AGES AT DEATH OF 541 CASES OF ALCOHOLISM.

	12–17	18–22	23–27	28–32	33–37	38–42	43–47	48–52	53–57	58–62	63–67	68–72	73–77	Over 78.	TOTAL.
Men	1	2	17	35	64	58	42	28	26	28	11	3	3	....	318
Percentage of Total	0.21	0.62	5.34	11.	20.12	18.24	13.52	8.8	8.13	8.8	3.45	0.94	0.94	....	
Women	1	6	29	34	42	43	13	24	11	13	4	3	....	....	223
Percentage of Total	0.45	2.69	13.	15.24	18.88	19.28	5.83	10.76	4.93	5.83	1.8	1.35	....	....	541

It is noticeable that the highest percentage of the men die in the same quinquennial period in which there are most admissions, while the highest percentage of female deaths is ten years later than the period of greatest admissions. Between 33 and 42 years both sexes show practically the same percentage—men, 38.4 per cent.; women, 38.2 per cent. But there is a very great difference in the number of deaths before 32 years of age, the percentages for the men being 17.2 per cent. and for the women 31.4 per cent., *i. e.*, nearly twice as many young women die of alcoholism as young men, and as far as the deaths from alcoholism are concerned men seem to live longer than women. The greater amount of muscular work that men are forced to do, by which the alcohol is burned up as a carbohydrate, thus exciting less drug action, would account for these facts. From the same cause there are both relatively and actually more fat alcoholic women than men. The bodies of the great majority of both sexes are well nourished, and this is striking when we consider for what long periods these patients subsist on alcohol alone without other food. It also shows well the food action of alcohol.

Beginning with the heart, the lesions found are very striking. In only 8 men and 2 women was the heart considered normal. Eleven men had the valvular lesions of chronic endocarditis, but no women. It is in the heart muscles that the lesions are found, showing that alcohol strikes at the functional contractile ability of the heart. Fatty degeneration is the most common lesion in both sexes (males, 28.8 per cent.; females, 42.8 per cent.), with fatty degeneration combined with brown atrophy (males, 25.5 per cent.; females, 17 per cent.) a close second. Brown atrophy is third and fibroid myocarditis fourth. And then there are various combinations of the above lesions, in only a few instances combined with fatty infiltration. These lesions occur at all ages, from the youngest to the oldest. It is especially interesting to note the frequency of brown atrophy of the heart muscle. This lesion is usually said to occur as a result of wasting diseases or from old age. It was present in the males in 36 hearts. One of these patients had carcinoma of the pylorus and pancreas. Eleven had tuberculosis, either active or healed, in the lungs or bronchial glands or both. In 3 old age—73, 76 and 77 years respectively—must be considered. So there remain 15, from 25 to 55 years of age, in whom alcohol may fairly be said to be the causative factor. Among the women it was present in 12 hearts. Four of these women had tuberculosis in bronchial nodes or lungs, leaving 8, 21 to 50 years of age, in whom the brown atrophy was present. There were also 21 males and 8 females showing tuberculous lesions, active or healed, in lungs or bronchial nodes, without cardiac brown atrophy and showing in the large majority of hearts only fatty degeneration. Ante-mortem thrombosis was present in some chamber of the heart in 5 men and 1 woman, and thrombosis in one of the coronary arteries was also found in 1 male.

The aorta and large vessels showed varying degrees of atheroma, most often of moderate degree, though not infrequently this lesion was very extensive, less often but slight. In but 6 men and 2 women are the vessels noted as normal. The lesion extended uniformly in aorta and vessels in most cases. In 5 cases it was the more extensive in the aorta; in 5 others the more extensive in the vessels. In 1 man the aorta and vessels showed but slight atheroma, while the coronary arteries were extensively calcified. Counting only the cases 50 years of age and under, we have 53 men, with extensive atheroma in 9, moderate in 26, slight in 14, none in 4. In 19 women we have extensive atheroma in 6, moderate in 6, slight in 6, none in 1.

The great frequency of atheroma is interesting from the fact that Lancereaux, in Paris, in his interesting lectures on alcoholism, states that atheroma of the aorta and vessels is so rare in alcoholics that its presence almost excludes alcoholism.

The most common conditions found in the lungs are œdema and congestion. Lobar pneumonia was present in 10 men and 3 women, broncho pneumonia in 14 men and 3 women, septic pneumonia in 1 man, and aspiration pneumonia in 1 woman, fibroid pneumonia in 6 men and 3 women. Active tuberculosis was present in 13 men and 1 woman, healed tuberculosis scars in 11 men and 2 women. Multiple emboli from a cardiac thrombus occurred in 1 man, and thrombosis of the pulmonary artery in 1 woman.

The liver shows more interesting lesions, as it is in this organ that the fatty degeneration and cirrhosis have always been designated as especially belonging to alcoholics. Until recently animal experimentation has not been able to prove this for fatty degeneration and has not yet proved it for cirrhosis. It has been found, however, by Rosenfeld, that fatty liver can be produced in dogs if these animals be starved for six days and then fed on alcohol. By this method, after more than four doses of 3½ to 4 cc. of

96 per cent. alcohol, the fatty content of the liver increases from the normal 10 per cent. found in starving dogs to an average of 22.6 per cent. If sugar be given with the alcohol the fatty degeneration does not take place. This would seem to indicate that the frequency of fatty degeneration in human beings may depend in some measure on the abstinence from food which is so common among alcoholics. In the 90 livers of the men examined, fatty degeneration alone occurred in 37 per cent.; in the women in 40 per cent.; in combination with cirrhosis, brown atrophy and parenchymatous degeneration, in 43 per cent. in men and 34 per cent. in women; *i. e.*, it was present in 80 per cent. of all the livers examined of the men and 74 per cent. in the women. In both sexes cirrhosis combined with fatty degeneration was the second most common lesion, being seen in 31 per cent. in the men and in 17 per cent. in the women. Cirrhosis was present in some degree in 48.8 per cent. in the men and in 34 per cent. in the women. The livers are designated as hypertrophic cirrhosis in but 3 cases of both sexes and atrophic cirrhosis in but 2 each. It is my belief, from clinical experience in the general wards of Bellevue Hospital, that the extreme degrees of cirrhosis do not so commonly occur as they did fifteen and twenty years ago. I have considered here as cirrhosis a visible increase in the connective tissue, whether the liver was larger or smaller than normal or of only normal size and weight. Formad, in Philadelphia, in 1886, found atrophic cirrhosis only 6 times in 250 post-mortem examinations on alcoholics. As I said above, the animal experimentation has not produced cirrhosis of the liver, but clinically we find a well-defined history of excessive alcoholic indulgence in the great majority of patients suffering from cirrhosis, and the occurrence of it here in over one-third of the women and in almost one-half of the men would certainly indicate its alcoholic origin. The method of its causation is still a disputed point. The pathologic action of alcohol as a cellular poison causing a degeneration and death of cells may be followed by an increase in the connective tissue and thus be the chief cause of cirrhosis, or since acute and chronic congestions are at times followed by an increased growth of the fibrous tissue, the cirrhosis may result from the chronic hyperæmia. Rosenfeld expresses with considerable reserve the view that cirrhosis of the liver is the result of very long continued alcoholic poisoning with doses which would cause most men to succumb. This he believes would explain the infrequency of cirrhosis relative to the large number of alcoholics.

It is interesting that the age at death of the male alcoholics with cirrhosis here recorded tends to support this view, the age of the men being in 13, 22 to 37, and from 48 to 77 in 31, the greatest number of deaths from alcoholism in men occurring between 33 and 37 years of age. In women the greatest number of deaths occur from 38 to 42. In the 12 cases of cirrhosis here given 6 are below and 6 above 40 years of age. In not a single patient of the 125 is the liver recorded as normal.

In the spleen chronic congestion was present in the men in 30 per cent. and in the women in 66 per cent., and fibrosis in 25 per cent. and 18 per cent. respectively.

In examining the lesions found in the pancreas the great frequency with which chronic fibrosis occurs is very noticeable. In the 74 times the pancreas is mentioned in the men chronic fibrosis occurs in 52 per cent., and in 28 of the women it occurred in 50 per cent. Fatty infiltration occurred in 16 per cent. of the men and in 14 per cent. of the women. The pancreas was normal in 23 per cent. of the men and in 25 per cent. of the women.

Fatty degeneration of the cortex of the adrenal bodies in 70 men was present in 34 per cent., and in 21 of the women in 57.5 per cent. These bodies were normal in 58.5 per cent. in the men and in 38.5 in the women.

In the kidneys there were always found some lesions; none are recorded as normal. The chronic lesions greatly predominate, acute nephritis being mentioned in only 2 men and 1 woman. Acute inflammation grafted on chronic was present in 7 men and 1 woman. The chronic diffuse nephritis with exudation—*i. e.*, chronic parenchymatous—often with congestion, was the most common, being present in the men in 50 per cent. and in 65 per cent. in the women. Chronic diffuse nephritis without exudation —*i. e.*, chronic interstitial nephritis—at times with congestion, occurred in 39 per cent. of the men and in 29 per cent. of the women.

The mucous membrane of the stomach showed chronic atrophic gastritis in 50 per cent. in the men and in 50 per cent. of the women. Hemorrhagic gastritis was present in 24 per cent. of the men and in 16 per cent. of the women. Sometimes these two conditions were combined.

The mucosa of the intestines was normal in about half the records of both sexes, and the most common condition varying from this was congestion, sometimes with œdema. One patient, a young man of 33, died of multiple emboli of the intestines, the origin of these emboli being a thrombus of the right auricle.

The bladder was ruptured in two men. It was normal in about half of both sexes, and in about 25 per cent. of both sexes there was either acute or chronic cystitis.

The ovaries in 12 women between 20 and 30 were 5 times markedly atrophic, and in women between 31 and 40 they were atrophic 5 times in 8. Thus, in 20 women under 40, in 50 per cent. they showed extensive atrophy. The testicles did not show any gross evidence of atrophy.

To classify accurately the lesions of the central nervous system is a difficult problem The brain was examined in 76 men and 29 women. Œdema and congestion of the membranes is almost always present, especially the œdema. Œdema of the cerebral tissue is mentioned as being present in 72 per cent. of the men and 51.7 per cent. of the women· Congestion of the tissues was present in 54 per cent. of the men and in 14 per cent. of the women. Pachymeningitis was present in 2 men and 3 women. Pachymeningitis hemorrhagica was present in 4 men and 2 women. Hemorrhagic meningitis was found in 3 men. Thickening, opacity and adhesions of the pia—*i. e.*, chronic meningitis—was observed in 65.8 per cent. of the men and in 41 per cent. of the women. Combined with this meningitis, sclerosis of the brain tissue with atrophy of the convolutions was observed in 31.5 per cent. of the men and 41 per cent. of the women. That atrophic degeneration of the brain substance is more frequent among women than men corresponds with one's clinical experience in treating alcoholics, for women seem to degenerate mentally more quickly and more completely from alcoholic excesses than men do. In no woman is the brain mentioned as normal, but in 1 man it is so described.

Combined with this cerebral sclerosis and its increased neuroglia there is an atrophy of the cerebral cells and a degeneration of the axis cylinders running out from them. Thus a descending degeneration to the cord occurs of which we will speak later.

Atheroma of the cerebral vessels is mentioned in 10 men and 7 women, but this is far below its actual occurrence. Miliary aneurism of cerebral vessels occurred in 4 men and 1 woman. Lesions of the brain from disease of the vessels is mentioned as follows: Recent cerebral hemorrhage, 2 men and 1 woman; old hemorrhage in 2 women, 1 hemorrhage being encysted in the pons; cerebral embolism in 1 woman; areas of cerebral softening in 6 men and 1 woman; 2 men and 2 women showed a sero-fibrinous exudate of acute meningitis; 1 man a septic meningitis and another a tubercular meningitis; 1 man showed an acute septic encephalitis.

The cerebral lesions so far spoken of are those of chronic alcoholism. In one young woman, 32 years of age, the lesions of the central nervous system in acute alcoholic degeneration were observed. This patient, previously an abstainer, sought oblivion and suicide by taking enormous amounts of alcohol. She died after a four days' poisoning. Her brain showed acute encephalitis, with marked cellular degeneration, with round cell infiltration along the blood vessels. Her spinal cord showed a degeneration of the posterior tracts and of the cells of the anterior horns. The peripheral nerves were normal. The spinal cord of several of those who showed cerebral sclerosis showed descending degeneration of the posterior tracts of the cord. In others the spinal cords were normal. In still others whose cords were apparently normal many degenerated fibres were seen in the posterior tracts when these cords were examined microscopically by Dr. Brooks. Weigert's stain does not bring this out, but a modification of Marchi's method shows them very distinctly. The congestion and œdema of the pia and dura mater of the brain were often continued in the membranes of the spinal cord, and the same was sometimes true of the chronic meningitis. In 5 women with peripheral neuritis (none of the men had this neuritis) in 2 there was ascending and descending degeneration of the tracts of the cord, in 2 only a descending degeneration, and in 1 there was only marked œdema and congestion, but no degeneration could be found after the most careful microscopic examination.

To sum up the pathology of chronic alcoholism, we have various degenerations of the viscera which, while not characteristic of the condition, are fairly constant in their occurrence. The hyperæmia, the cellular degeneration and the increase in the connective tissue in varying combinations make up the condition in the different individuals. We are dealing with a poison generated by a group of micro-organisms, the yeast fungi, and the resulting lesions are similar to those produced by bacterial poisons. The fatty degeneration of the heart muscle and other viscera is seen in the acute infectious diseases and the brown atrophy is seen in the chronic wasting of tuberculosis. Like bacterial toxins, alcohol is particularly a cellular poison; but because of its peculiar nature in its relation to the carbohydrates it exerts a double action of food and drug in the body. And because of the long continued dosage we see, besides the cellular effect, the chronic connective tissue changes which occur to replace the cell destruction, or as a result of the chronic hyperæmia. We are dealing, therefore, with patients whose vascular system is degenerated and who are suffering from a chronic poisoning of the nervous control of the vessels—*i. e.*, the vaso-motor centres—and with hearts whose contractile ability is seriously if not dangerously impaired. Their digestive organs, the pancreas, gastric mucosa, and even the intestinal mucosa are all functionally deranged and often show atrophy from fibroid infiltration. The chief source of chemical metabolism, the liver, is extensively degenerated, and the main avenue of elimination, the kidneys, are the seat of extensive acute and chronic inflammation and degeneration. What wonder is it, then, if we treat them as subjects of a nervous disease, forgetting the rest, we fail to aid them and see them die from cardiac and vascular asthenia, though we may deceive ourselves and think they died from delirium and serous meningitis.

The cerebral lesions are due to the direct poison of the alcohol, and also, in a large measure, to vascular atheroma; and many an alcoholic, as long as his heart and blood vessels and digestive apparatus hold out, lives long years with a degenerated brain, a nuisance and a burden to his community.

Alcoholics die from intercurrent infectious diseases, to which they are especially prone. They die from cerebral injuries caused by disease of the cerebral vessels. Many die suddenly from acute alcoholic poisoning of the cardiac muscle, especially those with fatty degeneration of the heart muscle, though this also occurs in those with normal hearts. The records of any public medical examiner will show this. A few die in whom the paralysis of the right heart predominates over that of the left, with marked congestion of the viscera and anæmic lungs. This occurred only once in the series here given. More die in whom the paralysis of the left heart predominates over that of the right, œdema and congestion of the lungs being an ante-mortem symptom.

Very many die from a paralysis of the vaso-motor system and failure of the heart as a whole, in whom the "wet brain," or serous meningitis, and gradual cardiac failure predominate in the clinical picture.

Individual idiosyncrasy is a marked feature in the resistance to alcohol, its tolerance and the lesions produced. Many men will consume without apparent effect amounts that will cause profound intoxication in others, or may even cause death. Old topers acquire a tolerance which seems remarkable when compared with those unaccustomed to the use of alcohol. The concentration in which the alcoholic fluid is taken and the physical amount of water with which the alcohol is diluted exert an influence on the effect produced.

The lesions produced vary greatly in different individuals; in some the circulatory apparatus shows the greatest intensity of the toxic effect; in others the liver is the most degenerated; and in others the central nervous system is the first to show the permanent lesions and give evidence of the most marked and progressive deterioration. In others there is for years no apparent lesion, and then some sudden strain on the organism occurs, its equilibrium is upset and the whole organism crumbles.

To study the clinical types in detail Dr. Theodore J. Abbott, one of my recent house physicians, has taken some 275 detailed histories. These show that the beginning of alcoholic indulgence occurred at a much younger age than one would suppose. In 259 instances where it was known, 4 began before 6 years of age, 13 between 6 and 12, 60 between 12 and 16, 102 between 16 and 21, 71 between 21 and 30, and 8 after 30 years of age. Thus over two-thirds—*i. e.*, 68 per cent.—began before 21 years of age. Almost all gave a history of intemperance in other members of their family. The reason for acquiring the habit is given as for the sake of sociability in 129; taught by their elders in 18; to be thought "tough" in 3; troubles, business or domestic, in 31; occupation, bartenders, icemen, etc., 18; to cure disease, as lumbago, pleurisy, lead colic, tic, etc., 23; out of work or on a vacation, 12; not known, 12.

The steady indulgence or periodicity of the spree was given as follows: First spree, 2; periodical spree, with abstinence between, 102; continuous indulgence, with heavy periodical spree, 70; chronic indulgence, drinking all they could get at every opportunity, 39; periodical sprees, with so-called moderate drinking in intervals, 47. The length of the spree for which they came to the hospital in 485 men was: Less than a week, 87, 17.9 per cent.; one to two weeks, 124, 25.5 per cent.; two to three weeks, 78, 16 per cent.; three to four weeks or longer, 196, 40 per cent.

These various types, with the "acute drunks," or those who had been drinking to excess for a few hours only, make up the alcoholic service in Bellevue. These last vary in number at different times and are usually in the minority. At times they are numerous, and then some line of treatment, such as washing out of the stomach or the use of hypodermatic injections, starts a rumor among the Bowery lodging-houses that "they are using the needle in Bellevue," and the acute cases drop off to almost none. The idea of the needle being that its use will take away the appetite for alcohol, and it is therefore to be avoided.

Few of the alcoholics drink because of the enjoyment of the taste, as the wine connoisseur does, and who rarely becomes a depraved drunkard. Most of them drink for the narcotic effect, either to obtain the feeling of well-being and indifference to their environment, or to seek oblivion, and like all narcotics, alcohol begets a craving for more. I believe that this craving is produced by the loss of tone in the small arteries and capillaries, because it disappears in both morphine and alcohol addictees when the vaso-motor tension is raised and kept up. If this is not done, then more narcotic is demanded to benumb the distressing depression which it produces.

The majority of the alcoholics among the men are whiskey drinkers, either this alone or with beer. The women, especially the young prostitutes, are the wine drinkers.

I have endeavored to learn if the cheap whiskeys, the compounded whiskeys, caused more tremor than the higher grades of the same fluid, thinking, at first, that the cheap whiskeys contained more fusel oil. But in this I was mistaken, as the analyses made by various observers seemed to show that the compounded whiskeys contained none, or less, than the genuine malt-distilled whiskeys. The tremors from the cheaper whiskeys are greater and last longer simply because, for the same amount of money, more whiskey can be obtained, and hence the poisoning is greater from the greater amount of ethyl alcohol consumed. From some recent events in New York it would seem that wood alcohol, methyl alcohol, is sometimes added to cheap whiskey. I do not remember seeing a single patient with sudden blindness, such as methyl alcohol gives, but from the tendency to fatal collapse certain acute cases have shown, and the rapidity of onset and furious delirium of other cases, I have long been struck with the intensity of toxic action that cheap whiskeys sometimes produce.

The tremor varies in the individuals from the variation of personal resistance and tolerance of the alcohol familiar to all of us in regard to all drugs. But if the tremor and the condition of the tongue be taken together one may make a fairly accurate guess as to the length of the spree. If the tongue is moderately coated and there is no tremor, it means a debauch of about five days; a heavily coated tongue and no tremor, about seven days; a moderately coated tongue and a slight tremor, about ten days; a heavily coated tongue and a marked tremor, about two weeks; a moderately coated tongue with a very severe tremor, about three weeks; a fairly clean tongue and a severe tremor, about a month; and a bright red, glazed tongue and a severe tremor, from six weeks to three months. The above is true of young men up to about 45 years of age. After that the tremor comes on earlier, and one must allow about twice as much tremor for the same length of spree—*i. e.*, a two weeks' spree will give as much tremor in an elderly man as a month's spree in a young man. In women these rules are useless; their nervous system is more intensely affected and is affected earlier. A week or ten days' spree brings, usually, an intense tremor, and it is very hard to obtain any accurate history from them. Female alcoholics do not seem to be able to tell the truth, even with assistance.

The symptoms of acute drunkenness are too familiar to need detailed description. The men are more sullen and calmly obstinate than the women, and more easily managed. The women are more ugly, vicious, abusive and obscene than the men. The pale, comatose " drunk " is in a more dangerous condition of low blood pressure and dilated vessels than the flushed face individual. One must never forget that acute drunkenness may hide some serious injury.

An acute debauch may produce a condition of such complete disassociation of ideas that the individual may be unable, when he comes out of this debauch, to remember any occurrence after the first few drinks. On the return of consciousness he may find a week or ten days has elapsed, and that he may be wandering about his own town, or he may even find himself in a strange city, many miles from where he started. During this time he may have appeared drunken and delirious or simply drunken.

A relatively short debauch may bring on a condition of alcoholic mania with the most vivid hallucinations, but this condition differs from true delirium tremens. In this maniacal condition the hallucinations produce no fear of the objects seen; they produce a vigorous, belligerent attitude toward them. The patient's attention can be distracted from them and he will explain logically his reasons for objecting to the acts of the imaginary persons who are annoying him or doing harm to his property. After a night's sleep produced by some hypnotic these patients wake up clear-headed, in excellent condition, and do not again become delirious. They usually have a clear memory of their hallucinations.

All alcoholics, at the end of a long spree, do not have true delirium tremens. They are sleepless, terrified (especially at night), tormented by evil dreams when they do sleep, and in a condition of pitiable collapse; but if fed and made to sleep they escape the real hallucinations. Others, while not actively delirious in the daytime, sink into a low muttering delirium at night, and, if not properly treated, may gradually sink into the condition of serous meningitis. This is more apt to occur in the elderly men. A prolonged spree is not necessary for the development of delirium tremens. It is not an uncommon occurrence to see men who, from their own statements and from those of their family and friends, have never been helplessly drunk, but have only taken alcohol continuously and in so-called moderate amounts, brought into the hospital having suffered a severe blow on the head, or a severe fall, or with a broken leg or arm, and yet develop delirium tremens. An acute infection may act in the same causal relation. These patients do not do well; they are apt to die in their delirium or to gradually sink into the " wet brain " stages and die.

The true delirium tremens may develop during a prolonged spree or in chronic alcoholics during an extra indulgence in their habitual amount of alcohol. It may come on several days after the cessation of a hard spree, and is then often ascribed by the patient to the fact that he left off alcohol and drank water. The hallucinations are usually connected with the daily occupation of the individual, but the animal or " menagerie " hallucinations are not uncommon. Snakes do not predominate so much as is popularly supposed. With these hallucinations there is a great fear and horror of them, and the intensity of the fear is a good criterion of the severity of the delirium. For instance, drivers usually are tending horses or driving them. As long as the horses go forward, stop or back according to the orders given them there is no bad prognostic significance, no matter how furiously active the driver may be; but if the horses back against his will, the terror inspired is distressing to witness. This form of delirium occurs only in the worst cases. The chief hallucinations are those of sight, those of hearing are next and those of touch third. Hallucinations of smell are rare.

The delirium usually clears up in a few days. Often, after it is gone in the daytime, it returns for a few nights. In very severe cases the delirium is ceaseless and causes an incessant muscular jactitation, which generates so much heat that the temperature rises rapidly and, unless controlled, may rise as high as 108 degrees F., and the patient usually dies in from twenty-four to thirty-six hours.

In summer the restlessness may cause a condition similar to heat stroke and the patients die quickly. Temperatures of 104 degrees or 105 degrees are of bad prognosis. Other severe cases show a continuous restlessness and delirium for several days and finally die exhausted, either suddenly or more slowly with the serous meningitis. Some cases do not become rational, and although their bodily condition improves, their mental condition does not, and they go on into alcoholic insanity, from which they may or may not recover after many weeks. The delirium tremens from beer does not come on as readily as that from whiskey, but is slower in clearing up. Many alcoholics, whether delirious or not, seem prone to epileptiform convulsions. Patients in whom these occur frequently give the history of having the convulsions only after they have been drinking and are free from them when abstinent. Patients who drink absinthe are especially prone to convulsions and to attacks of sudden loss of memory. They not infrequently show hyperæsthesia of the legs and complain of intense pain in the legs. This hyperæsthesia is said by Lancereaux to be characteristic of those who are addicted to the excessive abuse of the various liqueurs besides absinthe.

The treatment of alcoholics is usually routine, without much consideration of the individual; but consideration of the individual variations will give as much difference in successful results in this condition as in other diseases. In the mild acute intoxications a cold water shower is all that is necessary, and then rest and sleep. If they are without marked arterial changes, some emetic is advisable, either copper or zinc sulphate, and after the effects of this have subsided, grs. 5 of calomel and grs. 15 of soda bicarbonate, and if they are not inclined to sleep, 1 or 2 drachms of paraldehyde will be sufficient. When they awake, some milk and some capsicum, ginger and nux vomica as a stomachic is indicated. When the patients are maniacal or ugly, pugnacious and unmanageable, a hypodermatic injection of 1/10 grain of apo-morphine is the best treatment. It does not always cause emesis, but it rapidly changes the most pugnacious into a limp docility, followed quickly, on being put to bed, by a sleep of from two to three hours. The maniacal form of delirium will often resist all hypnotics given by mouth, but a hypodermatic injection of apo-morphine, gr. 1/10; hyocyamine, gr. 1/100, and strychnine, gr. 1/60 to 1/30, is the best to quiet them. Morphine alone will increase the cerebral congestion and should be avoided. Hyocyine, in my hands, has been most unsatisfactory; it seems to excite rather than soothe, especially in the women.

In treating the chronic alcoholics, the question most often discussed is that of the continuance or withdrawal of alcohol. I have tried both plans and am firmly convinced that, with the two exceptions to be mentioned later, alcohol should be immediately withdrawn and absolutely withheld. The heart and the vasomotor system must be vigorously stimulated; and if this is done, the recovery is quicker and more complete when alcohol is withheld than when it is given. The two drugs which give the best results under these conditions are strychnine and ergot used hypodermatically. The solution of ergot is that recommended by Livingston and is a drachm of the solid extract of ergot to one ounce of sterile water, with three minims of chloroform to preserve it, and lately Dr. Livingston has added three grains of chloretone to the ounce. The action of the ergot is to contract the unstriped muscular fibres of the vessels. It thus acts on the dilated vessels and brings the circulation nearer to its normal tone; it equalizes the circulation and reduces congestion. The equalization of the circulation reduces the strain on the heart and thus enables a degenerated heart to do its work with less effort. There may be also some stimulating action by ergot on the vasomotor centres which produces the same desired result. The exact physiologic action of ergot is as yet incompletely worked out; the results, however, obtained from its use are the equalization of the circulation, an increased tone in relaxed vessels, a prevention and an absorption of serous effusions and a reduction of congestion.

Besides the cardiac and vascular stimulation, rest, sleep and nourishment are the objects to be attained in treating delirium tremens and other forms of chronic alcoholism. When a patient comes under treatment during his spree and before he has made any endeavor to stop, often the quickest hypnotic is 2 drachms of paraldehyde in a half ounce of whiskey. This can safely be repeated at hour intervals for one or two doses. If, however, the patient has had no alcohol for twelve or twenty-four hours or longer, the paraldehyde should be given without the whiskey, because, in a small percentage of these patients the mixture of whiskey and paraldehyde seems to start up a delirium from which it takes several days to recover. Sometimes even the paraldehyde in the above doses will not cause sleep; then a mixture of chloral, gr. 15; morphine, gr. 1/8; tincture hyocyamus, drachm 1/2; tinct. zingeberis, m. 10; capsici, m. 3; in water, ounce 1/2, is effective. This also is safe to repeat after an hour's interval. The morphine in these amounts and with chloral does not have any deleterious effect. These two hypnotic doses have given me the most satisfactory results of the many I have tried. I do not hesitate to give 30 grains of chloral at a dose with 1/4 of a grain of morphine, and repeat, if necessary, in one or two hours.

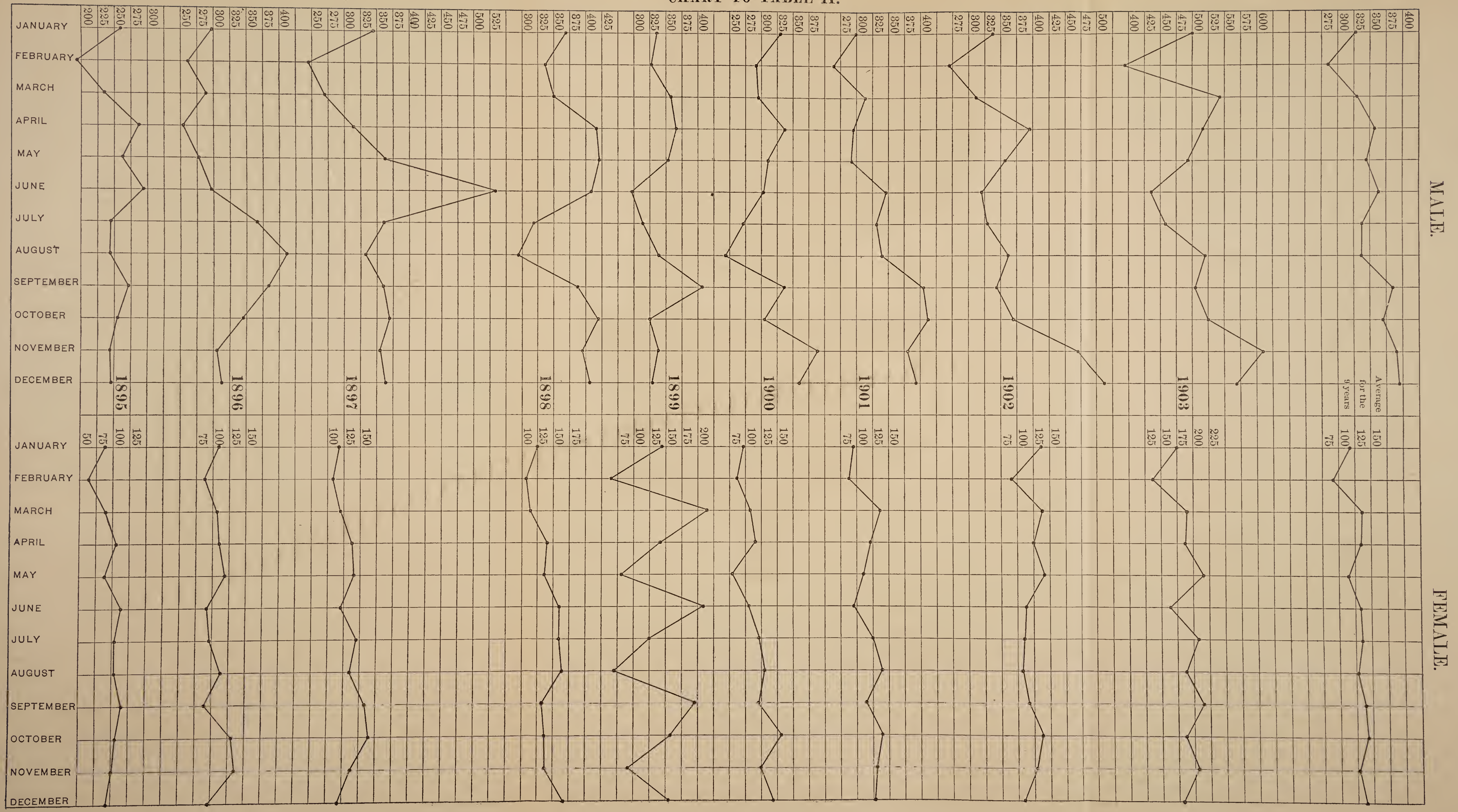
Small doses of hypnotics in delirium tremens are worse than useless. They are ineffective and the patient wears himself out by his ceaseless thrashing. Strychnine by hypodermatic injection, in 1/60 to 1/30 gr. doses, should be given at about four-hour intervals, and 30 minims of the ergot every two hours. As soon as a patient comes under treatment a cathartic should be given, and when he awakes from his sleep milk or eggs, and some bitter stomachic with capsicum aids his digestion and should be given with each feeding or only every four hours. Our usual mixture is tinct. capsicum, m. 2; tinct. nucis vomicæ, m. 6; tinct. zingiberis, m. 2; water up to a drachm. After a patient is over his delirium or has had a good sleep and is recovering, a mixture of tinct. nucis vomicæ, m. 10; tinct. cinchonæ co., m. 5; tinct. capsici, m. 2, and tinct. gentianæ co., ad drachm 1, is an excellent tonic mixture given every four hours. When a patient is convalescent the ergot injections may be given every four, six or eight hours, as the nervousness of the patient indicates. When a patient shows signs of serous meningitis by his low muttering delirium, apathy and stiff neck, the strychnine and ergot should be given day and night every two hours. The ergot here is more important than the strychnine. I have given the ergot every two hours for a week or ten days and have not yet seen any signs of ergotism and abscesses are not formed if it is properly given. It should be given straight into the muscles of the deltoid or glutei—not into the thigh in the vastus muscle.

Continued feeding with milk, broth and eggs is here very important. And after the patients are convalescing, sometimes egg nogs with a little whiskey seem to aid the digestion and absorption of their food. When patients are very weak and in collapse, aromatic spirits of ammonia and Hoffman's Anodyne are useful. Often liquor ammonii acetatis, ½ ounce, is given every four hours to the milder delirious patients and to those not delirious. Cold sprinkle baths are very useful in summer to quiet patients and to reduce the temperature caused by the muscular exertions. When necessary, patients should be restrained by sheets holding down their shoulders and by others restraining their feet. In very wild patients, bandages around their wrists and tied to the bed are at times necessary.

The mortality among the alcoholics in Bellevue during the past nine years has been 1,580 deaths in the 51,632, *i. e.*, 3 per cent. Among the males, 1,193 in 37,841, or 3.15 per cent.; among the females, 387 in 13,791, or 2.8 per cent. Among those whom I am certain have come under my personal charge the mortality in the 8,099 that I have treated without ergot has been 220 deaths, or 2.7 per cent.; 5,883 men, with 173 deaths, *i. e.*, 2.9 per cent.; 2,216 women, 47 deaths, *i. e.*, 2.1 per cent. Among 1,995 patients that I have treated with ergot there were 31 deaths, *i. e.*, 1.5 per cent.; men, 1,537, 24 deaths, *i. e.*, 1.5 per cent.; women, 458, 7 deaths, *i. e.*, 1.5 per cent. That is an improvement by the use of ergot of 44 per cent. in the death rate. This has been brought about by the diminished number of patients who have suffered from serous meningitis and by the increased number who have recovered when this condition developed. Formerly I lost 98 per cent. of the cases of serous meningitis. With ergot I have lost about 40 per cent. The ergot reduces the tremor rapidly and in a marked degree, so much so that in those who have received three or four injections I can no longer tell by the tremor and tongue the length of their spree. It aids very materially the action of the hypnotics, so that a diminished dosage of these is required. It quiets the delirium, so that a much greater number of the delirious patients will rest quietly in bed without restraint. It reduces the gastric congestion and thus improves the appetite.

It annuls the fearful depression and nervousness following the debauch and withdrawal of alcohol. It prevents, in many cases, the paralysis of the vaso-motors and relaxation of the vessels, and thus prevents the serous meningitis. It will, in a certain percentage, cause a reabsorption of the serous-meningeal exudate and thus cure the " wet brain." Ergot is the only drug I have yet found which will accomplish this. It is no new remedy, nor magic potion, but an old remedy newly applied—so old-fashioned that it has been almost discarded and is still discredited. If one uses it properly and fearlessly, and remembers that in delirium tremens and chronic alcoholism it is not in the brain that the danger lies, but that it is in the degenerated heart muscle and diseased and disorganized circulation, one will save many a patient that formerly was beyond assistance.

CHART TO TABLE II.



CURVES OF MONTHLY ADMISSIONS IN ALCOHOLIC WARDS.

1895-1903.

CHART I.

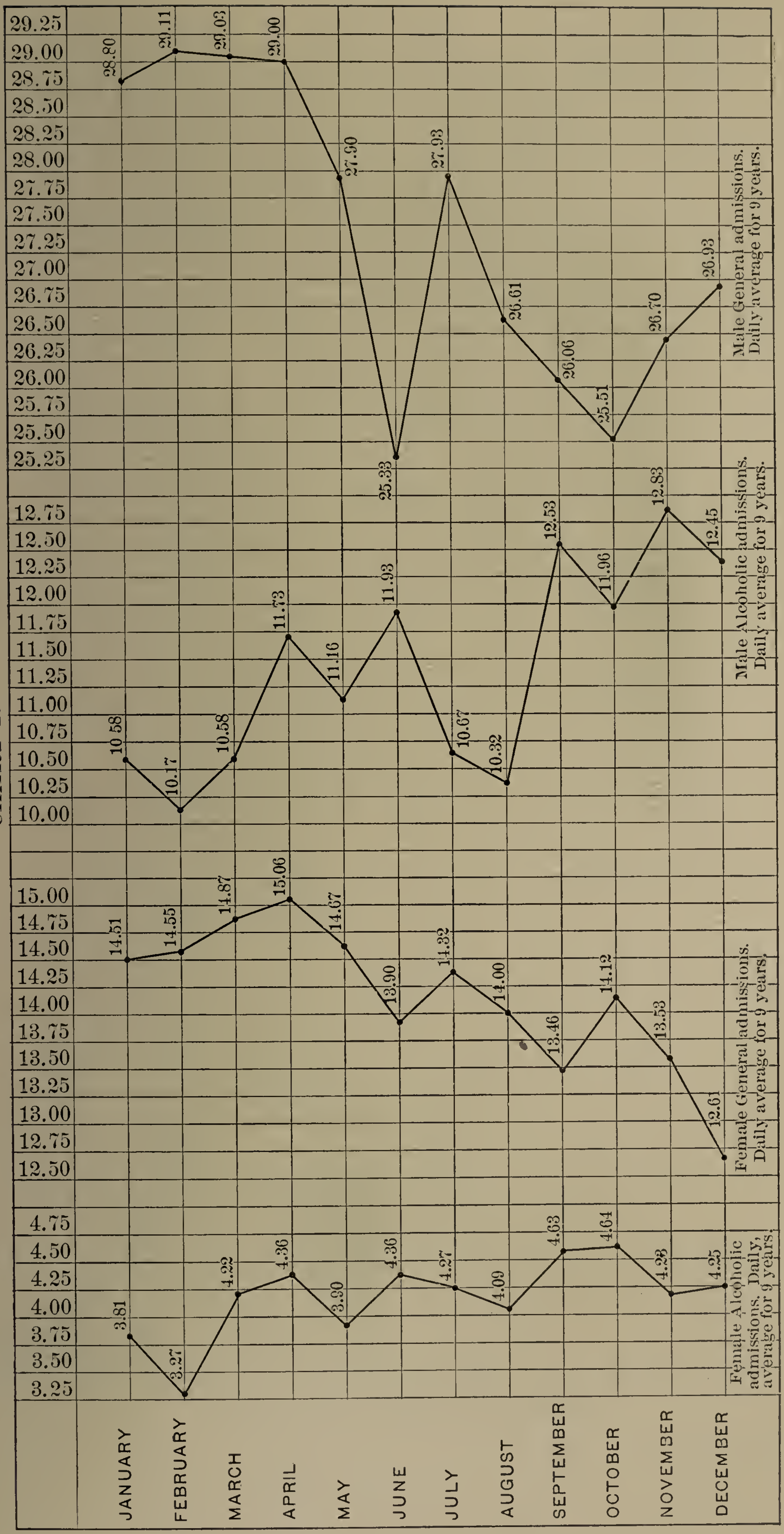
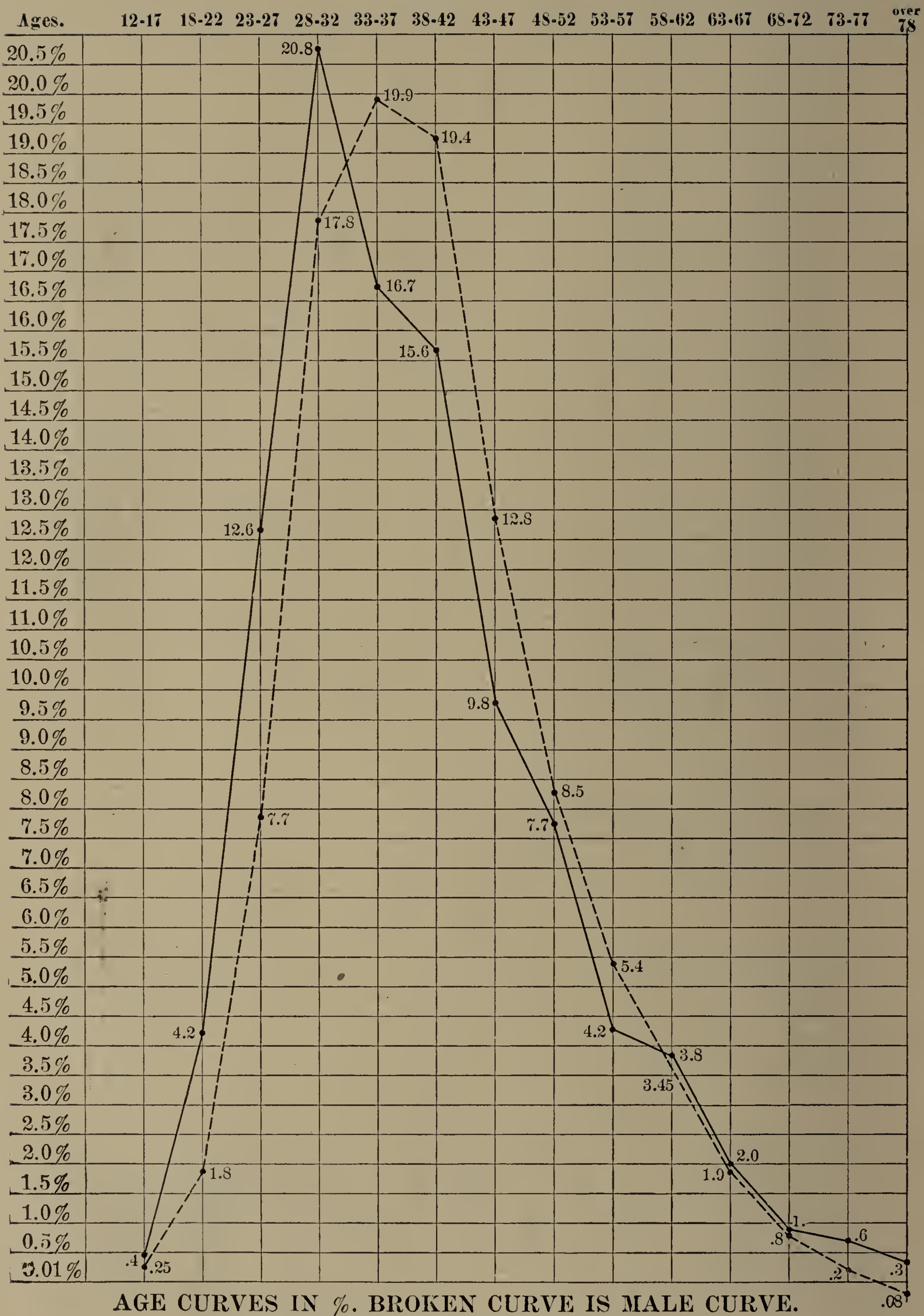

29.25
29.00
28.75
28.50
28.25
28.00
27.75
27.50
27.25
27.00
26.75
26.50
26.25
26.00
25.75
25.50
25.25
28.80
29.11
29.03
29.00
27.90
25.33
27.93
26.61
26.06
25.51
26.70
26.93
Male General admissions.
Daily average for 9 years.
12.75
12.50
12.25
12.00
11.75
11.50
11.25
11.00
10.75
10.50
10.25
10.00
10.58
10.17
10.58
11.73
11.16
11.93
10.67
10.32
12.53
11.96
12.83
12.45
Male Alcoholic admissions.
Daily average for 9 years.
15.00
14.75
14.50
14.25
14.00
13.75
13.50
13.25
13.00
12.75
12.50
14.51
14.55
14.87
15.06
14.67
13.90
14.32
14.00
13.46
14.12
13.53
12.61
Female General admissions.
Daily average for 9 years.
4.75
4.50
4.25
4.00
3.75
3.50
3.25
3.81
3.27
4.22
4.36
3.90
4.36
4.27
4.09
4.63
4.64
4.23
4.25
Female Alcoholic admissions. Daily, average for 9 years.
JANUARY
FEBRUARY
MARCH
APRIL
MAY
JUNE
JULY
AUGUST
SEPTEMBER
OCTOBER
NOVEMBER
DECEMBER

CHART III



AGE CURVES IN %. BROKEN CURVE IS MALE CURVE.
UNBROKEN IS FEMALE CURVE.

TABLE II.

	1895.		1896.		1897.		1898.		1899.		1900.		1901.		1902.		1903.		TOTAL 9 YEARS.		AVERAGE 9 YEARS.		DAILY AVERAGE 9 YEARS.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
January.	258	85	298	105	340	112	369	125	332	144	326	99	300	94	335	132	501	174	3,059	1,070	328	118	10.58	3.81
February....	195	67	260	83	242	106	333	103	326	62	294	87	258	88	272	96	395	140	2,585	832	287	92	10.17	3.27
March	230	88	286	103	262	115	348	112	359	215	298	106	314	138	318	131	542	191	3,057	1,199	328	131	10.58	4.22
April........	288	102	251	105	318	130	415	141	366	145	331	118	293	124	389	125	523	191	3,174	1,181	352	131	11.73	4.36
May	263	89	276	116	356	132	417	133	354	76	309	77	290	112	356	136	498	218	3,119	1,089	346	121	11.16	3.90
June.	290	117	293	93	527	122	409	163	297	206	301	103	348	94	324	114	438	169	3,227	1,181	358	131	11.93	4.36
July	245	102	362	96	356	145	316	160	316	125	271	121	330	124	326	112	458	207	2,980	1,192	331	132	10.67	4.27
August......	245	102	406	117	328	130	292	162	336	73	248	126	341	133	360	110	522	194	2,978	1,147	330	127	10.32	4.09
September...	272	109	380	92	355	151	380	129	406	197	336	124	410	116	344	120	502	219	3,385	1,257	276	139	12.53	4.63
October.	255	103	347	126	368	156	417	134	325	162	304	155	419	139	372	134	525	195	3.332	1,304	370	144	11.96	4.64
November...	243	101	304	129	352	136	392	135	336	88	381	122	380	103	471	127	607	206	3,466	1,145	385	127	12.83	4.23
December....	247	94	311	99	355	120	403	165	328	173	357	139	395	101	514	112	569	186	3,479	1,194	386	132	12.45	4.25
Totals...	3,031	1,159	3,774	1,264	4,159	1,555	4,491	1,660	4,081	1,671	3,756	1,377	4,088	1,366	4,381	1,449	6.080	2,290	37,841	13,791	...	...	.....	....

DISTRIBUTION BY AGES.

CLASSES.	OCCUPATIONS.	AGES BY QUINQUENNIAL PERIODS.														Totals.	RATIO PER 1,000	Total in Manhattan and Bronx by U.S. Census, 1900.
		12–17	18–22	23–27	28–32	33–37	38–42	43–47	48–52	53–57	58–62	63–67	68–72	73–77	78 and over.			
Professional	Architects, artists, teachers of art, designers, draughtsmen, etc.	...	1	2	5	7	6	5	5	2	1	...	...	...	...	34	7.6	4,468
	Clergymen, priests	...	...	...	...	...	2	...	...	...	1	...	...	...	...	3	1.7	1,698
	Engineers, surveyors, electricians	...	3	7	15	16	16	1	3	...	...	...	...	...	...	61	9.5	6,427
	Journalists, reporters	...	1	6	15	21	23	8	11	5	1	1	...	...	1	93	56.1	1,647
	Lawyers	...	...	2	6	5	7	4	5	1	...	1	...	...	...	31	6.	5,160
	Musicians and teachers of music	...	...	2	11	6	8	4	1	1	...	2	...	...	...	35	7.6	4,606
	Physicians and surgeons	...	...	1	9	12	9	6	5	4	...	...	...	...	...	46	9.9	4,617
	School teachers	...	...	...	1	...	2	1	...	...	1	1	1	...	...	7	2.9	2,419
	Actors	...	...	5	16	17	15	11	1	3	2	2	2	...	...	74	21.9	3,372
	Chemists, dentists, students and others of this class	6	2	7	6	10	6	5	5	1	1	1	...	...	...	50	....	......
Commercial and official	Agents, auctioneers and collectors	...	...	6	20	29	29	17	7	7	6	2	2	1	...	126	9.6	13,186
	Bookkeepers, clerks, accountants, etc.	1	12	72	180	162	164	112	80	37	18	10	1	...	...	849	14.	60,520
	Bankers, brokers and officials higher than clerks	...	...	3	6	14	15	11	5	2	3	1	...	...	...	60	9.3	6,475
	Stenographers and typewriters	...	3	8	15	10	2	2	1	...	...	1	...	...	...	42	24.	1,745
	Others of this class	3	1	1	1	3	4	2	1	...	1	1	...	...	...	18	....	.. ...

Mercantile	Apothecaries and pharmacists	...	...	...	7	5	2	1	1	1	2	...	...	...	...	19	....	......
	Commercial travelers and salesmen	...	8	9	57	75	89	67	32	19	13	4	2	1	...	376	12.8	28,552
	Merchants and dealers	...	...	4	9	13	23	21	19	4	3	...	...	...	...	96	2.1	45,244
	Hucksters and peddlers	...	1	6	14	9	28	13	14	12	7	2	1	...	1	108	10.9	9,850
	Others of this class	1	...	3	2	7	7	2	1	6	4	2	...	...	...	35	....	......
Public entertainment	Hotel and boarding-house keepers	...	...	1	2	1	3	1	...	2	1	...	...	...	...	11	6.2	1,769
	Bartenders, liquor dealers, saloon and restaurant keepers	...	6	34	76	95	72	30	13	6	1	1	...	...	...	334	25.5	13,114
	Waiters	...	11	46	93	84	72	34	19	17	8	3	3	...	...	390	*24.8	*24,794
Personal service	Barbers and hairdressers	...	...	...	12	11	7	7	8	3	...	2	...	1	...	51	6.6	7,702
	Janitors, undertakers and sextons	...	1	7	8	7	5	9	3	9	11	1	2	...	...	63	11.8	5,334
	Policemen, watchmen detectives	...	...	2	9	10	8	6	7	6	6	1	1	...	1	57	....	......
	U. S. soldiers, sailors and marines	1	...	4	3	9	4	2	...	3	...	...	1	...	...	27	2.6	10,182
	Nurses and hospital orderlies	...	...	7	14	18	26	4	4	4	2	...	...	...	...	79	72.8	1,084
Laborers, not agricultural, and servants	Laborers	2	51	196	415	434	506	308	210	125	95	38	14	10	3	2,407	35.3	68,031
	Servants	...	3	12	23	49	49	33	24	16	9	6	2	...	...	226	*24.8	*24,794
Manufacturing and mechanical trades.	Bakers and confectioners	...	...	4	13	12	14	22	16	5	7	3	1	...	...	97	10.4	9,291
	Blacksmiths and horseshoers	...	2	12	16	19	22	11	11	5	3	8	1	...	...	110	28.5	3,860
	Boot and shoe makers	1	...	2	2	5	5	8	7	16	7	8	2	...	...	63	9.5	6,594
	Brewers, distillers, spirit rectifiers, etc.	...	...	1	...	...	2	...	2	3	...	1	...	...	...	9	5.8	1,557
	Butchers	...	...	4	14	14	15	22	10	11	6	7	1	...	...	104	11.6	8,911
	Cabinet makers and upholsterers	...	1	3	7	2	14	7	5	5	1	1	...	...	...	46	10.8	4,225
	Carpenters and joiners	...	1	7	20	26	34	24	24	12	8	5	3	...	...	164	10.8	15,093
	Cigar makers and tobacco workers	...	1	1	6	5	5	10	4	4	1	2	...	...	...	39	4.4	8,946
	Clock and watch repairers and jewelers	...	1	1	...	...	1	6	2	3	1	1	1	...	...	17	21.5	789

* † Calculated together as one occupation.

Classes.	Occupations.	Ages by Quinquennial Periods.														Totals.	Ratio Per 1,000	Total in Manhattan and Bronx by U.S. Census, 1900.
		12–17	18–22	23–27	28–32	33–37	38–42	43–47	48–52	53–57	58–62	63–67	68–72	73–77	78 and over.			
Manufacturing and mechanical trades.	Compositors, printers and pressmen..	...	6	12	41	60	35	28	17	6	3	2	1	...	...	211	20.2	10,404
	Coopers..............................	...	...	...	2	5	3	1	4	2	...	...	...	...	...	17	42.5	400
	Engineers and firemen, not locomotive	...	...	15	37	50	52	33	30	23	16	4	2	...	...	262	29.4	8,891
	Glassblowers and workers...........	...	1	1	...	2	...	...	...	...	...	...	...	...	...	4	5.9	679
	Hat and cap makers.	...	...	1	2	4	3	1	3	2	...	...	...	...	...	16	7.4	2,163
	Iron and steel-workers, boiler-makers.	...	2	12	33	31	40	23	16	15	3	5	1	...	...	181	31.9	5,680
	Leather-makers......................	...	...	...	3	...	2	1	1	...	...	...	...	...	...	7	11.9	585
	Leather-workers.	...	...	...	5	6	6	5	5	3	1	2	...	...	...	33	19.	1,736
	Machinists and mechanics...........	...	2	11	25	29	22	18	5	6	5	1	2	...	...	126	17.3	7,258
	Marble and stone-cutters............	...	1	3	13	14	13	16	12	4	1	1	...	...	...	78	25.	3,157
	Masons, brick and stone..............	...	1	13	46	28	19	25	12	8	3	...	...	...	1	156	19.4	8,047
	Mill and factory operatives (textile)...	...	1	2	5	5	6	2	1	1	...	...	...	...	...	23	8.2	2,786
	Millers, flour and grist..............	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.....	....	94
	Painters, glaziers and varnishers......	...	...	17	30	76	78	59	37	21	14	4	3	1	...	340	20.5	16,558
	Plasterers, lathers and whitewashers.	...	...	1	11	14	14	7	7	2	1	...	2	1	...	60	22.3	2,689
	Plumbers, gas and steam-fitters......	1	9	14	41	33	25	19	4	7	3	1	...	...	...	157	15.1	10,335
	Tailors..............................	...	2	7	16	21	24	17	6	8	9	6	1	...	...	117	2.5	42,751
	Tinners, roofers, coppersmiths.......	...	2	8	14	11	14	7	5	3	2	...	1	...	...	67	19.4	3,453
	Others of this class..................	1	2	29	51	75	57	46	29	16	9	6	3	...	...	324	....	......

Agricultural, transportation and other outdoor.....	Boatmen and canal-men..............	...	...	...	7	3	4	10	3	1	4	2	1	1	...	31	†51.2	†3,648
	Drivers, hackmen, teamsters, expressmen, ice men, etc..........	1	27	111	207	211	142	93	52	38	12	15	7	1	...	917	27.9	32,431
	Farmers..........................	...	...	1	2	1	2	3	...	1	...	2	...	...	...	12	38.6	313
	Gardeners and florists...............	...	1	1	1	5	5	1	2	1	...	1	...	...	...	18	17.4	1,030
	Horse trainers, livery stable keepers, hostlers, grooms, jockeys, etc.	...	1	11	42	31	27	25	14	3	6	1	1	...	...	162	38.3	4,224
	Lumbermen	...	...	3	...	...	1	2	1	...	...	1	...	...	...	8	114.3	70
	Miners and quarrymen..............	...	...	...	3	1	1	1	...	1	...	...	...	...	...	7	26.7	262
	Sailors, pilots, fishermen and oystermen....	...	2	8	19	29	25	20	19	8	11	6	2	...	2	151	†51.2	†3,648
	Steam and street R.R. employees.....	...	2	7	19	17	25	6	6	4	...	...	...	...	...	86	7.7	11,057
	Stockmen, drovers, etc........... ..	...	...	...	...	...	...	...	1	...	...	...	...	...	...	1	15.8	63
	Others of this class...	...	1	...	...	7	4	2	...	...	...	...	...	...	...	14	....	......
	Other occupations, telegraph operators, etc.......................	...	2	5	21	17	17	8	7	1	1	...	...	...	...	79	....	......
	No occupation given..................	9	16	41	78	113	72	41	34	24	32	27	19	3	...	509	....	......
	Totals........................	27	191	822	1,902	2,121	2,059	1,357	899	569	367	205	87	20	9	10,636	....	......

* † Calculated together as one occupation.

DISTRIBUTION BY NATIONALITIES.

CLASSES.	OCCUPATION.	NATIONALITIES.										Other Nationalities.	TOTAL.
		United States.	Ireland.	Germany.	England and Wales.	Scotland.	Scandinavian.	Canada.	Italy.	France.	Russia.		
Professional	Architects, artists, teachers of art, designers, draughtsmen, etc.	12	7	2	8	1	..	2	..	..	..	2	34
	Clergymen, priests	..	1	1	1	..	..	..	..	..	..	..	3
	Engineers, surveyors, electricians	44	10	4	2	..	..	1	..	..	..	..	61
	Journalists, reporters	61	9	2	10	8	..	1	..	2	..	..	93
	Lawyers	27	2	2	..	..	..	..	..	..	..	..	31
	Musicians, teachers of music	19	3	4	3	1	..	5	..	..	..	..	35
	Physicians and surgeons	30	6	2	1	2	1	4	..	..	..	..	46
	School teachers	2	2	..	..	..	..	2	..	1	..	..	7
	Actors	58	8	1	7	..	..	..	..	..	..	..	74
	Chemists, dentists, students and others of this class	36	2	3	2	1	1	..	1	..	2	2	50
Commercial and official	Agents, auctioneers and collectors	72	23	6	12	2	2	7	..	..	1	1	126
	Bookkeepers, clerks, accountants, etc.	561	172	36	37	15	4	19	..	..	2	3	849
	Bankers, brokers and officials higher than clerks	42	10	5	..	1	..	..	..	..	..	2	60
	Stenographers and typewriters	30	5	..	3	2	..	2	..	..	..	..	42
	Others of this class	6	3	..	3	1	1	3	..	..	1	..	18

Mercantile	Apothecaries and pharmacists	13	2	3	..	..	..	1	..	..	..	..	19
	Commercial travelers and salesmen	238	67	12	25	10	5	13	..	1	2	3	376
	Merchants and dealers	56	17	7	2	..	1	2	..	..	..	1	96
	Hucksters and peddlers	48	27	22	6	1	..	..	1	..	2	1	108
	Others of this class	22	7	1	2	..	1	..	1	1	..	..	35
Public entertainment	Hotel and boarding-house keepers	4	4	2	..	..	1	..	..	..	..	..	11
	Bartenders, liquor dealers, saloon keepers and restaurant keepers	167	93	48	13	..	1	1	1	1	1	3	334
	Waiters	145	166	24	27	12	4	6	1	2	..	3	390
Personal service	Barbers and hairdressers	23	6	13	1	1	..	1	2	1	2	1	51
	Janitors, undertakers and sextons	37	17	6	1	..	2	..	..	..	..	..	63
	Policemen, watchmen, detectives	33	17	3	2	2	..	..	..	..	..	..	57
	U. S. soldiers, sailors and marines	21	5	1	..	..	..	..	..	..	..	..	27
	Nurses and hospital orderlies	38	30	3	3	3	2	..	..	..	..	..	79
Laborers, not agricultural, and servants	Laborers	985	1,155	97	51	18	24	15	22	5	5	30	2,407
	Servants	86	54	31	23	3	5	3	1	9	..	11	226
Manufacturing and mechanical trades	Bakers and confectioners	19	24	41	5	3	..	..	..	2	..	3	97
	Blacksmiths and horseshoers	33	61	5	5	5	..	..	1	..	..	..	110
	Boot and shoe makers	11	27	19	1	1	1	2	..	..	..	1	63
	Brewers, distillers and spirit rectifiers	1	2	5	1	..	..	..	..	..	..	..	9
	Butchers	43	24	31	1	1	..	..	..	1	1	2	104
	Cabinet makers and upholsterers	22	7	8	1	3	1	..	..	2	..	2	46
	Carpenters and joiners	78	49	10	5	4	9	4	1	1	1	2	164
	Cigar makers and tobacco workers	15	..	16	1	..	..	..	..	..	1	6	39
	Clock and watch repairers and jewelers	6	..	7	1	1	..	..	..	..	..	2	17
	Compositors, printers and pressmen	159	29	7	4	4	..	6	1	1	..	..	211

CLASSES.	OCCUPATIONS.	NATIONALITIES.										Other Nationalities.	TOTAL.
		United States.	Ireland.	Germany.	England and Wales.	Scotland.	Scandinavian.	Canada.	Italy.	France.	Russia.		
Manufacturing and mechanical trades	Coopers	8	6	2	..	..	..	..	..	..	1	..	17
	Engineers and firemen, not locomotive	139	107	4	7	..	..	2	..	..	..	3	262
	Glass blowers and workers	2	1	..	..	1	..	..	..	..	..	..	4
	Hat and cap makers	11	4	1	..	..	..	..	..	..	..	..	16
	Iron and steel workers, boiler makers	105	51	6	6	6	4	1	..	..	..	2	181
	Leather makers, tanners and curriers	2	2	..	2	..	..	..	..	..	1	..	7
	Leather workers, trunk makers, etc., harness makers	12	15	2	2	..	..	..	..	..	1	1	33
	Machinists and mechanics	77	23	12	6	2	2	1	1	1	1	..	126
	Marble and stone cutters	34	29	8	4	..	..	1	..	..	1	1	78
	Masons (brick and stone)	98	42	5	10	..	..	1	..	..	..	..	156
	Mill and factory operatives (textile)	10	8	2	2	..	..	..	..	1	..	..	23
	Millers (flour and grist)	..	..	..	..	..	..	..	..	..	..	..	....
	Painters, glaziers and varnishers	212	61	37	13	4	3	4	..	1	2	3	340
	Plasterers, lathers and whitewashers	33	14	1	10	2	..	..	..	..	..	..	60
	Plumbers, gas and steam fitters	122	26	2	3	3	..	1	..	..	..	..	157
	Tailors	33	46	12	7	1	3	2	..	1	7	5	117
	Tinners, tinware roofers, coppersmiths	45	12	7	..	..	2	..	..	..	..	1	67
	Others of this class	210	42	28	18	4	7	7	..	2	..	6	324

Agricultural, transportation and other outdoor.........	Boatmen and canalmen	19	14	3	..	..	..	..	..	..	..	..	36
	Drivers, hackmen, teamsters, expressmen, icemen, etc..........	559	295	27	22	5	1	1	1	1	..	5	917
	Farmers..................	5	2	1	..	..	1	..	1	..	..	2	12
	Gardeners and nurserymen and florists........................	10	3	4	1	..	..	..	..	..	..	..	18
	Horse trainers, livery stable keepers, hostlers, grooms, jockeys, etc......	56	90	5	5	2	..	1	..	..	1	2	162
	Lumbermen	4	4	..	..	..	..	..	..	..	..	..	8
	Miners and quarrymen..	6	..	..	..	..	..	1	..	..	..	..	7
	Sailors, pilots, fishermen and oystermen.....................	72	34	9	12	5	11	3	..	1	..	4	151
	Steam and street railroad employees.........................	39	39	3	1	3	..	..	..	..	..	1	86
	Stockmen, drovers, etc......................................	1	..	..	..	..	..	..	..	..	..	..	1
	Others of this class...	10	3	..	..	1	..	..	..	..	..	..	14
	Other occupations, telegraph operators, etc..................	61	5	6	2	..	..	3	..	..	1	1	79
	No occupation given..	259	137	40	20	7	3	9	7	5	8	14	509
	Totals...	5,567	3,273	717	423	152	103	138	43	43	45	132	10,636

STATISTICS OF WOMEN, SHOWING AGE, NATIONALITY AND OCCUPATION.

OCCUPATIONS.	12–17	18–22	23–27	28–32	33–37	38–42	43–47	48–52	53–57	58–62	63–67	68–72	73–77	Over 78.	Total.
Housework and domestic servants	2	46	165	333	282	275	150	132	79	60	40	14	7	5	1.590
Housewife	....	10	39	87	78	54	28	20	8	7	5	2	2	....	340
Milliners, dressmakers	....	4	11	18	17	42	23	15	7	7	5	1	2	....	152
Cigarmakers, factory hands, etc	1	3	11	9	11	4	2	6	2	....	....	....	....	....	49
Bookkeepers, clerks, stenographers, etc	....	4	15	19	11	2	....	1	1	....	1	....	1	....	55
Scrub women, hospital helpers, etc	....	....	3	7	9	9	10	6	4	3	4	....	2	....	57
Miscellaneous	....	1	9	14	6	5	....	3	....	....	2	1	1	1	43
No occupation given	29	274	777	1,208	948	885	582	443	241	234	105	68	35	17	5.846
	32	342	1,030	1,695	1,362	1,276	795	626	342	311	162	86	50	23	8,132
Irish	1	75	314	683	599	591	409	382	235	230	112	58	25	16	3,730
Canada and United States	18	187	591	786	573	469	267	167	83	47	18	13	7	5	3,231
English	....	15	31	84	91	100	35	41	8	8	4	1	1	....	419
German	2	15	34	46	24	38	21	9	9	8	9	2	3	1	221
Scotch	....	1	19	34	28	22	20	12	9	4	6	4	1	....	160
Other nationalities	9	43	29	51	55	54	37	19	8	10	6	2	3	1	328
	30	336	1,018	1,684	1,370	1,274	789	630	352	307	155	80	40	23	8,089
	IRISH.	U. S. AND CANADA	ENGLISH.	GERMAN.	SCOTCH.	TOTAL.									
Housework and domestic servants	883	561	63	44	39	1,590	....	....	....	....	....	....	....	....	....
Housewife	142	154	24	17	3	340	....	....	....	....	....	....	....	....	....
Seamstresses, dressmakers, milliners, tailoresses	56	81	10	3	2	152	....	....	....	....	....	....	....	....	....
Cigarmakers, factory hands, etc	12	32	2	....	3	49	....	....	....	...	....	....	....	....	....
Bookkeepers, clerks, stenographers, etc	9	43	3	....	....	55	....	....	....	....	....	....	....	....	....
Scrub women, hospital helpers, etc	30	20	3	....	4	57	....	....	....	....	....	....	....	....	....
Miscellaneous occupations	9	25	6	2	1	43	....	....	....	....	....	....	....	....	....

"PERNICIOUS" ANÆMIA.

Notes on Fourteen Cases.

By W. Gilman Thompson, M. D.

The following series of fourteen cases of "pernicious" anæmia is reported on account of the comparative rarity of the disease and the obscurity of the idiopathic form of anæmia. The cases have all been observed in the Second Medical Division during the past four years, for the most part in the service of the writer, although one or two overlapped into that of Drs. Dana, Loomis and Coleman. The blood examinations were made by the staff of the Clinical Laboratory of the Cornell University Medical College. I am indebted also to Dr. W. L. Niles, late House Physician on the Division, for valuable data regarding several of the cases. Eleven cases, including seven men and four women, are presented with fairly complete clinical records. One patient was discharged "cured," one "unimproved," two were "improved" and lost sight of, and one is still under treatment. Nine patients died in hospital; in three cases no autopsies were obtained; in six autopsies were performed, and in three microscopic examinations also were made of the viscera.

Cases 10, 11 and 12 comprise autopsy records only. Prior to the installation in the hospital of the present admirable system of history filing, many histories were lost or buried in the mass of records, and, unfortunately, for these three autopsies corresponding clinical memoranda have not been discovered by diligent search.

Numbers 13 and 14 refer to cases in which the anæmia presented many of the "pernicious" characteristics, but the origin was clearly secondary to other maladies.

Case I was that of a blacksmith, a Swede, 44 years of age. He was a moderate drinker, non-syphilitic. No etiology could be discovered, and the only previous illnesses which the patient recalled were an attack of pleurisy and several attacks of gout. About five months prior to admission to the hospital, and without assignable cause, the patient became gradually pale, weak and short of breath on exertion. His hands and feet began to swell, and he complained of persistent frontal headache, palpitation, faintness after exertion, constipation and attacks of vomiting accompanied by epigastric pain.

Upon examination the patient appeared very pale, with flabby muscles, but without loss of tissue. Tophi were observed in the ears and great toes. The arteries were much thickened. The heart was normal in size, and at the apex both a systolic and presystolic murmur were heard; the pulse was small, feeble and rapid. There was evidence of slight congestion at the base of both lungs, and the legs were moderately œdematous. The abdominal viscera, as well as all reflexes and sensory phenomena appeared normal. There were no hemorrhages at any time. Examination of the blood showed 2,024,000 red corpuscles, and the differential count of white cells was as follows:

Small mononuclear lymphocytes, 32 per cent.
Large mononuclear lymphocytes and transitional forms, 9.6 per cent.
Polynuclear neutrophiles, 56 per cent.
Eosinophiles, 1.7 per cent.

The patient remained a fortnight in hospital, when, having improved considerably, he went home and was lost from further observation.

CASE 2 was that of an Irish housewife, 55 years of age. The patient's mother died of hemorrhage of some sort, otherwise the family history was negative. There was no alcoholic or venereal history, and the patient's health had been good prior to six months before admission to the hospital, when she began to suffer from vomiting, anemia, moderate jaundice and œdema of the feet. Her appetite and bowels remained normal, but she lost weight and became very feeble. The œdema continued.

On admission, the patient appeared of a lemon yellow color, and the general nutrition was very poor. The heart was slightly enlarged to the left, and a mitral regurgitant murmur was heard, with an accentuated pulmonic second sound. The arteries appeared normal. Liver dullness extended two inches below the free border of the ribs; the spleen was normal; there were no enlarged glands; the knee-jerks were diminished, but there were no other nerve symptoms; the urine was normal; the temperature was 103 degrees F., but fell to 100 degrees F. the day after admission. The blood examination was recorded as follows:

Red cells, 848,000.
White cells, 2,500.
Hæmoglobin, 18 per cent.
Color index, 1.1.
Polynuclear cells, 61 per cent.
Lymphocytes, 31.75 per cent.
Large mononuclear, 7 per cent.
Eosinophiles, 0.25 per cent.

Four hundred cells counted showed 5 nucleated red cells, 2 normoblasts and 3 of intermediate form. There was marked poikilocytosis, polychromatophilia and basophilia.

After five days in hospital the patient insisted upon returning to her home, when the case passed from further observation.

Case 3.—The notes of the following case have been kindly furnished by Dr. Walter L. Niles, in whose service in the Out-Patient Department of the Hospital, Second Medical Division, the patient is still undergoing treatment (January, 1905).

The patient is an Irish housewife, 37 years old. The family history is negative. There is no venereal or alcoholic history, and the patient has had no children or miscarriages. She claimed to have had "malaria" fourteen years ago, but otherwise enjoyed good health up to one year ago (January, 1904), when, without definite cause, she lost strength and became gradually very pale. For several months previously the patient had had frequent attacks of morning vomiting, occurring about every other day. She had nausea and occasional headache, but until recently no other symptoms, and there was no hemorrhage of any sort. Quite recently the patient has complained of puffy eyelids, but has had no œdema elsewhere. There has also developed marked dyspnœa on exertion. Urine normal; appetite fair, despite the vomiting; no nervous symptoms.

On examination, the patient appeared fairly nourished and the skin showed marked white pallor. The pulse was small, soft and compressible and slightly increased in rate (90); no heart murmurs were audible. The lungs and other viscera all appeared normal, except the spleen, which appeared slightly enlarged, but not palpable. There was no œdema and no glandular enlargement.

The following six blood examinations were made, with differential counts based upon 500 cells:

Date.	Red cells.	White cells.	Hæmoglobin.	Color Index.	Polymorphonuclear.	Lymphocytes.	Large mononuclear.	Eosinophiles.	Mast cells	Megaloblasts.	Normoblasts.
			%		%	%	%	%			
1904 Dec. 2	2,400,000	2,800	70	1.5	57.7	29.2	10.6	2.	0.5	1	0
" 9	1,872,000	4,400	50	1.3	54.4	31.6	13.	1.	0.	1	4
" 16	2,056,000	4,000	50	1.2	57.4	29.2	13.2	0.	0.2	3	14
" 23	2,408,000	3,000	60	1.25	57.5	29.2	12.2	1.	0	0	8
" 30	2,424,000	3,000	68	1.4	56.	27.2	14.2	2.2	0.2	0	1
1905 Jan. 6	2,224,000	5,000	83	1.8	56.7	21.	20.	1.75	0.5	0	0

In degrees varying from "marked" to "slight," the following conditions were observed: anisocytosis, poikilocytosis, polychromatophilia, granular basophilia. In one specimen were many macrocytes.

The treatment has been dietetic and hygienic with administration of iron and arsenic, and during the six weeks of treatment to date there has been considerable improvement in the patient's general condition.

Case 4 was that of an Irish laborer 49 years of age. No etiology for the anæmia could be discovered. The man had been a hard drinker up to his forty-fourth year, when, having acquired both syphilis and gonorrhœa, he became alarmed about his habits and ceased drinking to excess. Without assignable cause, some months previous to his admission to the Hospital, he found himself gradually becoming very weak, and was finally obliged to stop work on that account. He had suffered from headache, but had no other complaint. On admission he showed decided pallor, but was without emaciation, although his muscles were extermely soft. There was slight enlargement of the inguinal and axillary glands. The heart was normal in size, and a faint blowing systolic murmur was heard over the apex, and there was decided pulsation in the vessels of the neck. The arteries appeared normal. All the abdominal viscera were normal. The blood exhibited the typical characteristics of advanced pernicious anæmia. The reflexes and sensory phenomena were normal throughout. During the three months that the patient remained under observation he became progressively weaker; the temperature ranged between 98 degrees and 100.4 degrees F., and the pulse, which was very feeble, varied from 86 to 112; there was occasional vomiting. There was no œdema, dyspnœa or hemorrhage. Toward the end of life the red blood corpuscles fell to 1,000,000, and the hæmoglobin to 22 per cent., color index 0.9, the white cells remaining normal in number. At the end of three months the patient died of asthenia and, unfortunately, no autopsy could be obtained.

Case 5 was that of an Austrian male, a cigarmaker, 52 years of age. For two years prior to admission to my service the patient suffered from pains in the knees and swelling of the ankles, and for six months he had shown increasing pallor, with occasional vomiting. Otherwise the patient's health had been always good, and he gave no evidence of syphilis or alcoholism. On admission he appeared ill-nourished and extremely pale. The heart, spleen and other viscera appeared normal, but the knee-jerks were slightly increased. The urine showed a trace of albumin, with hyaline and granular casts. The temperature varied for days between normal and 101 degrees F. The patient's vomiting was continuous and uncontrollable, and he soon became very weak, stuporous and delirious. After three and one-half weeks in hospital he died of exhaustion, and no autopsy could be obtained. Five blood examinations were made as follows, only two of which were complete:

	Jan. 29, 1900.	Feb. 2, 1900.	Feb. 4, 1900.	Feb. 10, 1900.	Feb. 19, 1900.
Red blood cells........	1,488,000	1,776,000	1,958,000	1,909,000	1,828,000
White blood cells......	4,000	3,400	6,000	5,800	1,200
Haemoglobin..........	40 per ct.	40 per ct.	45 per ct.	62 per ct.	45 per ct.
Color index	1.3	1.1	1.1	....	....
Polymorphonuclear	59.5 per ct.	59 per ct.	....	....	....
Lymphocytes..........	30.9 per ct.	30.4 per ct.	....	....	....
Large mononuclear.....	7 per ct.	8.5 per ct.	....	....	....
Eosinophiles...........	1.7 per ct.	1.9 per ct.	....	....	....
Mast cells.............	.7	0	....	....	....
Normoblast............	1.	0	....	....	....

The red cells were large and poikilocytosis was marked.

Case 6.—The patient was a German, male, aged 47 years; a moderate drinker, with a negative venereal history. For many years he had chewed at least three ounces of tobacco a week, which might account in part for his gastric and nerve symptoms. There was no etiology discoverable for the patient's anæmic condition; his health had been uniformly good until three years prior to admission to the hospital, when he suffered from an attack of anæmia, gastralgia and jaundice after eating improper food. From this attack he recovered and was well until three months before admission, when he gradually became very pale, weak and short of breath on working. Marked tremor of the hands and numbness in the hands and feet with formication appeared. The patient soon became unable to pick up small objects, or hold a glass of water, on account of tremor. He complained also of vertigo on rising and difficulty in getting his bearings in walking. There was no hemorrhage and no vomiting.

Upon examination, the patient appeared of a pale lemon-yellow color, without emaciation, but with œdema of the face and feet. The heart was normal in size and a soft systolic hæmic murmur was heard over the apex. The vessels of the neck presented a bruit and pulsation, the radial pulse was soft and rapid and the arteries were markedly sclerosed. Enlarged lymph glands were found on the right side of the neck, and a supernumerary nipple was observed on the right breast. The liver was slightly enlarged and the other viscera appeared normal. The nerve symptoms were of special interest, and were only partially explainable through tobacco poisoning. They were as follows: feeble left knee-jerk; absent right knee-jerk; slight ataxia of legs and hands; slight intention tremor, especially of left hand, with moderate paræsthesia and anæsthesia of that hand and of the toes; pupils small, the right larger than the left; they responded to accommodation, but not to light; both optic discs were pale. There was a purulent chronic otitis media of the right ear.

During several weeks, while in hospital, the pulse averaged 99-100, and the temperature rose to 101.4 on several occasions, reaching 104° F. two days before death, which occurred from asthenia, three months after admission to the hospital. On admission the blood examination by Dr. Hastings was reported as follows:

Red cells, 1,400,000.
White cells, 6,000.
Hæmoglobin, 25 per cent.
Polynuclear, 40 per cent.
Large mononuclear, 25 per cent.
Small mononuclear, 35 per cent.
Eosinophiles, 0.

Among 300 cells counted were one normoblast and two gigantoblasts; stained specimens showed also marked polychromatophilia and fresh specimens exhibited mycrocytes, macrocytes and poikilocytes. Many red cells showed degeneration areas. Coagulation time, four and one-half minutes. Three months later, and the day before the patient's death, the blood examination showed:

Red cells, 1,008,000.
White cells, 7,000.
Hæmoglobin, 20 per cent.
Normoblasts, 4 (300 cells counted).
Gigantoblasts, 7 (300 cells counted).

Other cells as in previous examination.

No autopsy was obtained.

Case 7 was that of an Irish housewife, 66 years of age. The family and previous personal history was negative. The patient was well up to five months before admission to the hospital, when she had an attack of grippe, which left her in a feeble condition, from which she did not recover. Three months prior to admission she complained of dyspepsia and vomiting without apparent cause. She lost weight and strength rapidly and suffered from headache. These symptoms persisted until admission.

On admission, the patient was emaciated, weak and very pale; the muscles were soft and the skin was dry and yellowish. The sclerotics were slightly yellow. There was moderate arteriosclerosis. The heart was somewhat enlarged towards the left and a soft blowing systolic murmur was heard over the apex and base. The other viscera appeared normal. The temperature varied between 99.6 degrees and 100.6 degrees F., and the pulse, which was feeble, ranged from 95 to 115. There was slight œdema of the ankles and legs. The reflexes and sensory symptoms were normal. The blood examination showed—

Red cells, 1,084,000.
White cells, 1,500.
Hæmoglobin, 25 per cent.

A differential count was not recorded.

There were no hemorrhages at any time. A gastric analysis showed absence of both hydrochloric and lactic acid.

After three weeks the patient developed a large abscess over the upper jaw, which extended to the bone, and having failed to respond to any treatment, she died.

Autopsy.—The autopsy data have been kindly furnished for this report by Dr. James Ewing, who made the post-mortem examination.

Body emaciated, extremely anæmic, slightly yellowish, few subcutaneous hemorrhages.

Heart.—Size normal, considerable fibrous thickening of all the valves. No stenosis or insufficiency. Muscles very fatty. Few sub-endocardial hemorrhages. Blood reduced in quantity, very watery, does not coagulate.

Lungs.—Extremely emphysematous, much ante-mortem œdema.

Liver.—Size normal, surface and section smooth, of peculiar dark-brown color, some fibrous peri-hepatitis. Gall-bladder distended, ducts pervious.

Femur.—Red marrow extends throughout right femur.

Spleen.—Organ small, capsule wrinkled, weight 4 ounces, section uniformly red; small supernumerary spleen.

Thyroid.—Normal.

Kidney.—Slightly reduced in size, cortex thin, numerous small cysts, markings obliterated, capsule not adherent.

Stomach.—Capacity seems moderately reduced, wall contracted at pyloric region and thinned especially over fundus; mucosa seems markedly atrophic, except at pyloric end; no glands to be seen by naked eye.

Intestine.—Entire length of small intestine thickly coated with bile-stained mucus. Mucous membrance of small intestine seems normal; Peyer's patches normal; no fæcal matter in small intestine.

Colon.—Seat of numerous small submucous hemorrhages.

Genital organs atrophic.

Brain.—Excessive œdema of pia, which is spotted with numerous small hemorrhages. Dura mater considerably and uniformly thickened. Cord looks normal.

Anatomical Diagnosis.—Atrophic gastritis, progressive pernicious anæmia, chronic diffuse nephritis, fatty heart, emphysema, extensive hyperplasia of red marrow.

Microscopic Examination.

Heart.—Shows extreme fatty degeneration and increase of pigment in muscle cells.

Lungs.—There is much œdema and marked dilatation of many air-vesicles.

Liver.—The outer two-thirds of lobules shows an extensive deposit of hemosiderin. Remainder of lobules holds considerable yellowish pigment not blackening with ammonium sulphide. The capillaries are irregularly dilated, and the cells in such areas are slightly atrophic.

Spleen.—Malphigian bodies are intact. There is moderate congestion of sinuses and the pulp cords contain an excess of lymphocytes, and many pigmented macrophages lie over the cords.

Marrow.—In the mid-femoral region there is nearly complete atrophy of fat cells, owing to hyperplasia of large cells with large vesicular and compact nuclei. Myelocytes and megaloblasts are about equally abundant and there are many islands of 30 to 50 normoblasts. There are many lymphocytes uniformly distributed.

Kidney.—There is a diffuse growth of old connective tissue throughout the cortex. The capsules of the glomeruli are often thickened, and the large arteries show fibrous thickening of all coats. The tubule cells are slightly degenerated and some tubules are dilated occasionally forming small cysts. Adrenals negative.

Pancreas.—Exhibits following lesions:

(1) Moderate chronic interstitial pancreatitis. Islands of Langerhans normal.

(2) Considerable fat invasion replacing the pancreatic tissue.

(3) Adenomatoid hyperplasia of epithelial lining of many ducts.

(4) Chronic fibrous thickening of many septa holding ducts.

Gastro-intestinal Tract.—The gastric mucosa is nearly everywhere digested down to an hypertrophied muscularis mucosæ. In some preserved areas there is a marked excess of lymphocytes and some strands of new connective tissue distorting the glands. At the pylorus the mucosa is intact and here are seen stages of advanced chronic interstitial productive gastritis, the glands being extensively replaced by fibrous tissue with many lymphocytes. At the pylorus the muscularis is the seat of extensive fibrosis.

Ileum.—Preservation good. In a few foci of miliary size there is advanced chronic productive inflammation of mucosa. Everywhere the thickness of the mucosa is reduced and the glands of Lieberkühn are shallow.

CASE 8 was that of a Swiss housewife, 49 years of age. The family and previous history of the patient was unimportant. She was a multipara, having had five children. Six years before admission to the hospital the patient was ill for three months with typhoid fever, but subsequently recovered completely. There was no venereal history and the menopause occurred in the forty-seventh year. Three weeks before admission the patient became weak and complained of vertigo, and of numbness in the hands, fingers and toes. There was also loss of power in the arms and a tendency to let fall anything held in the hands. A month earlier her friends had told her she looked yellow, but she felt well at that time. There was slight dyspnœa on exertion and occasional cough. Digestion was normal, and the bowels were not constipated. On admission, the patient appeared stout, but was very pale and somewhat jaundiced. The skin was very dry, and the mucous membranes were blanched. The patient appeared greatly exhausted and the pulse was feeble and quick (100). A soft blowing systolic murmur was heard over the pulmonic valve, and there was visible pulsation in the neck. The lungs were normal except slight dullness and a few pleuritic râles at the right apex. The abdominal viscera appeared normal, except the spleen, which was enlarged to the extent of an inch below the ninth rib. The knee-jerk was exaggerated on both sides, and the epigastric reflex was absent. Other reflexes were normal. There was no œdema except at the ankles. The urine was normal. The patient remained in hospital nearly four months, during which time ten examinations of the blood were made, showing progressive improvement. The first examination, made on admission, and one made three months later, are herein compared (the counts were made by the late Dr. J. Metcalfe Polk and by Dr. T. W. Hastings).

	ADMISSION.	THREE MONTHS LATER.
Red cells....	1,280,000	3,760,000
White cells......................	3,500	5,000
Hæmoglobin......................	30%	70%
Color index......................	1.2	·99
Polynuclear.	61.2%	54.6%
Small mononuclear...............	32%	33%
Large "	6%	11.2%
Eosinophiles......................	0.6%	1%
Mast cells	0.2%	0.2%
Normoblasts..................	12	2
Megaloblasts......................	6	0
....	500 cells counted.	500 cells counted.

In the second of these counts the red blood cells were found well developed, and there were only a few granular degenerated erythrocytes. In the various intermediate counts a slowly progressive improvement was demonstrated:

The polynuclear cells ranged between 61.2 and 34 per cent., the large mononuclear between 16 and 6 per cent., the small mononuclear between 53 and 28 per cent. The coagulation time, when the blood was at its poorest, was 7 minutes. During the patient's stay in hospital her temperature frequently rose to 100 degrees F., and on three or four days to 101 degrees F. While in hospital the patient improved in all her symptoms, under treatment with Fowler's solution, iron diet, rest and fresh air, so that after four months she insisted upon returning to her home. Two months later she returned to the hospital, complaining of all her original symptoms, especially of great weakness, abdominal pains and rectal pain. Her blood at this time showed marked deterioration, and an examination by Dr. Hastings was reported as follows:

Red cells, 1,790,000.
White cells, 2,400.
Hæmoglobin, 38 per cent.
Color index, 1.
Polymorphonuclear, 41 per cent.
Lymphocytes, 46.1 per cent.
Large mononuclear, 9.3 per cent.
Eosinophiles, 0.6 per cent.
Mast cells and myelocytes, 0.

Three hundred cells were counted and among them 5 normoblasts and 4 megaloblasts were found, with a few macrocytes, and marked polychromatophilia and granular basophilia. The patient remained in the hospital for two months, becoming rapidly weaker, and, failing to respond to any treatment, she died of asthenia. There was no hemorrhage at any time. A final blood examination, made two days before death, gave the following result:

Red cells, 856,000.
White cells, 9,000.
Hæmoglobin, 10 per cent.
Color index, 0.6.
Polymorphonuclear cells, 61.2 per cent.
Lymphocytes, 29 per cent.
Large mononuclear, 9.5 per cent.
Eosinophiles, 0.2 per cent.
No mast cells or myelocytes.

Four hundred cells were counted, among which were 25 normoblasts and 10 megaloblasts. There was extreme poikilocytosis, a few macrocytes were found, and, as in the previous examination, there was marked polychromatophilia and granular basophilia.

Autopsy.—The record of the autopsy has been kindly given to me for this report by Dr. James Ewing, who performed the post-mortem examination:

Body poorly nourished, very anemic, skin faintly jaundiced, moderate general anasarca.

Heart.—Valves, chambers normal, muscle distinctly fatty. No distinct hypertrophy. Blood deficient in volume, feebly coagulated in right chambers, watery.

Lungs.—Congested, œdematous.

Liver.—Size normal, consistence reduced, organ has general appearance of chronic congestion and recent fatty degeneration, but outlines of lobules are indistinct and the fatty points are irregularly distributed throughout the lobules.

Spleen.—Enlarged, rather firm, section brownish red.

Kidneys.—Size normal. Capsule adherent in places, cortex irregular in thickness, markings indistinct but regular.

Bone-marrow.—There is firm reddish lymphoid marrow throughout femoral shaft.

Gastro-intestinal Tract.—Stomach is contracted and empty. The pylorus admits a lead pencil with difficulty and the muscularis here seems thicker than normal. The mucosa is digested in places, elsewhere the *état mamellonée* is distinct. The small intestine contains a little light colored fecal matter and mucus in which are flecks of blood. The mucosa seems normal. Peyer's patches are atrophic.

Anatomical Diagnosis.—Pernicious anæmia, chronic gastritis, fatty heart, fatty liver, chronic interstitial nephritis, chronic hyperplastic splenitis, congestion and œdema of lungs, lymphoid hyperplasia of marrow, atrophy of Peyer's patches, general anasarca.

Microscopical examination of viscera.

Heart.—There is well-marked fatty degeneration and much increase in the pigment about the nuclei of the muscle cells.

Lungs.—Negative.

Liver.—The parenchyma cells are in a state of intense granular and fatty degeneration. Very numerous focal necroses are distributed throughout the organ, sometimes located about the central veins, but also found in other portions of the lobules. Many of the foci are infiltrated with blood. Brownish yellow pigment grains are abundant in many cells irregularly distributed.

Kidney.—There is moderate acute granular degeneration of tubule cells and considerable deposits of hemosiderin. The arterioles show moderate fibrous endarteritis. The cells of the glomeruli are increased in number and the lobules of the tufts are often fused together. The glomerular capsules are slightly thickened and the stroma of the cortex is increased in a few areas.

Spleen.—The Malpighian bodies are diminished in size. The sinuses are gorged with blood and contain many macrophages inclosing red cells and their detritus. Eosinophile cells are extremely abundant.

Lymph-Nodes.—In the retroperitoneal and mesenteric nodes there is moderate fibrosis and diminution in bulk of nodules and pulp-cords.

Bone-marrow.—In the marrow of the vertebræ there is extensive cellular hyperplasia with obliteration of the sinuses. Lymphocytes are deficient in number, neutrophile myelocytes are in excess, and eosinophiles are very numerous. Megaloblasts and islands of normoblasts are extremely abundant and these cells are the chief factors in the cellular hyperplasia. The marrow is therefore rather typical of the Cohnheim-Rindfleisch variety of medullary lesion, but an unusual proportion of the nucleated red cells are of nearly normal size.

Gastro-intestinal Tract.—The gastric mucosa exhibits a well-marked increase of fibrous tissue between the glands and a pronounced uniform excess of lymphocytes. The glandular layer is therefore thickened and the glands distorted, but they do not appear to be reduced in functional capacity. The lesion is that of a comparatively early stage of an active and progressive chronic catarrhal and interstitial gastritis. There is interstitial myositis at the pyloric orifice. Brunner's glands are intact.

The ileum shows a poor development of Peyer's patches, but no other changes.

Pancreas.—There is slight interstitial pancreatitis.

CASE 9 was that of a German bricklayer, 38 years of age. The family history was negative. The patient was a moderate drinker of beer and whiskey. Venereal history was negative. For half a dozen years the patient had attacks of nocturnal epilepsy, in which he would bite the tongue; and the following day experience headache and vertigo. The patient was married and had one boy. Ten months before admission the patient had severe headache, which lasted for two months, but he was otherwise well and was stout. Six months before admission the patient noticed sensations of numbness, prickling and coldness, which began in his toes and later affected the fingers. The legs became ataxic and later there was an umbilical girdle sensation. He also had abnormal gastric sensations; he became very pale and began to emaciate. He came to the hospital for relief of pains in the legs, but, having delusions of persecution, he was at first taken to the Insane Pavilion. He had had diarrhœa for three weeks.

On admission to the hospital the patient appeared very weak and emaciated. The skin was dry and of a pale lemon-yellow color. The legs were stiff and cold. There was moderate enlargement of the inguinal, epitrochlear and cervical lymph glands. The arterial walls were much thickened. A faint systolic murmur was heard over the apex of the heart, and a loud hum in the cervical veins. The liver was enlarged and extended an inch below the margin of the ribs. The other viscera appeared normal. The muscles of the left leg and thigh were atrophic.

The knee-jerks (especially the left) were exaggerated and the toes gave the Babinski reaction, but the other reflexes, as well as pain and tactile sense, appeared normal. While in hospital the patient for several months retained marked delusions of hearing and of persecution and poisoning by the nurses, etc., refusing at times both food and medicines. He was irritable and apathetic. The general nerve symptoms became worse; the legs were very weak, and legs, hands and arms became more markedly ataxic; the abdominal reflex was absent, but the cremasteric persisted; the finger tips lost their tactile acuteness; there was obstinate constipation and difficulty in urinating. The eyes appeared normal and the pupils reacted to light and distance. The temperature was irregular; at times normal, again rising to 100 degrees to 101 degrees F. The pulse averaged 80 to 90. The urine was normal. There were no hemorrhages at any time. The feces were searched for ova and parasites with negative result.

The patient, who was admitted August 14, 1902, made slow improvement under treatment by diet, Fowler's solution, iron and hypodermic injections of sodium cacodylate. His mental condition improved and his strength so far increased that he was able to sit in a chair. After four and a half months, early in January, 1903, he suddenly became very much worse, the hæmoglobin fell to 10 per cent. and the red cells to 550,000, and he seemed upon the verge of death. Without any change in treatment, however, he as suddenly began to improve in nutrition and color, and within four weeks, by February 3, 1903, the hæmoglobin rose to 40 per cent. and the red cells to 1,667,000. The improvement lasted for several months, when the patient again became very weak and died of asthenia on September 12, 1903, having been almost thirteen months under observation in the hospital. The apparently spontaneous variations in the blood composition are so interesting that they are appended in full. Many examinations were made by members of the Clinical Laboratory Staff of the Cornell University Medical College, chiefly by Dr. T. W. Hastings, and also by Dr. John W. Coe and the late Dr. J. Metcalfe Polk. These examinations vary somewhat in completeness, for in three of them differential counts were not made.

Date.	R. B. C.	W.B.C.	Hæmoglobin.	Polynuclear.	Lymphocytes.	Large mononuclear.	Eosinophiles.	Mast cells.	Normoblasts.	Megaloblasts.	Color Index.
1902			%	%	%	%	%				
Aug. 11	862,000	4,780	20	..	..	..	..	..	4	1	..
" 23	....	....	..	73	40	4.2	1.5	0.3	1	0	..
Sept. 3	648,000	3,600	25	54	31	13	2	..	..	4	..
" 12	....	.....	..	70.3	23	5.3	1	0	0	0	..
Oct. 2	960,000	10,000	45	..	..	..	..	..	..	..	..
" 11	864,000	8,500	30	62	35.2	2	0.8	0	38	0	1.5
" 28	1,102,000	6,000	40	..	..	..	..	..	..	..	..
1903 Jan. 10	550,000	4,000	10	55	35	10	0	0	few	+	..
" 22	1,197,000	6,300	28	72	22	4.3	4	0	1	+	1.2
Feb. 3	1,667,000	6,700	40	63	26	6.6	1.8	2.4%	3	0	1.2
" 12	1,642,000	5,000	40	58.8	31	7	2.6	0.2%	2	few	1.2

Autopsy performed by Dr. Otto H. Schultze:

Left Lung.—Chronic adhesions. Upper lobe very pale and emphysematous. Marked ecchymoses on lower surface. Lower lobe œdematous and atalectatic.

Right Lung.—Greater part of upper lobe and almost entire middle lobe markedly emphysematous and pale. Posterior surface of pleura shows dilated lymphatics very distinctly. At apex is a large fibrous and caseous nodule. Posterior portions very œdematous. Pericardium contains one ounce of serum.

Heart.—Right side markedly and left ventricle moderately distended with feebly clotted blood. Aorta normal in calibre, moderate atheroma. Aortic cusps slightly thickened at attachments. Mitral valve normal. Wall of left ventricle 1½ cm. thick, distinctly pale and opaque. Coronary arteries normal. Right ventricle 7 mm. thick. Tricuspid and pulmonic valves normal. Weight 18 ounces.

Peritoneum and omentum normal. Mesentery and abdominal fat normal.

Spleen.—Smooth, weight 5 ounces, section normal.

Liver.—Vessels at hilus negative, weight 4 pounds 6 ounces, section light brownish red, markings very obscure.

Pancreas normal.

Kidneys.—Slightly larger than normal, weight 8 ounces, capsule slightly adherent, section extremely pale, cortex increased in width, both ureters distended, adrenals normal, bladder greatly distended with urine.

Stomach.—Mucosa normal, of grayish tint at pylorus, duodenum bile-stained.

Colon.—At hepatic flexure are several areas where mucosa is raised and the summits of the folds necrotic, similar areas are scattered throughout colon, rectum normal, small intestine normal, prostate normal.

Skull.—In left parietal bone there is a defect lying 7 cm. from the coronal suture and 5 cm. from the longitudinal sinus. In it lies a foreign body, black, gritty, 2 cm. long, ½ cm. wide, pointed end. This body is surrounded by connective tissue, pierces the dura and enters cortex of brain causing a distinct depression ½ cm. wide and ½ cm. deep, located at junction of gyrus supramarginalis and parietal cortex. Cord appears normal.

Marrow of mid femur purely fatty. At epiphyses, red marrow appears in patches only. In ribs the marrow is diffluent.

Anatomical Diagnosis.—Pernicious anæmia, old pulmonary tuberculous focus, œdema of lungs, chronic pleurisy, chronic diffuse nephritis, ulcerative colitis, pigmentation of liver, deficient lymphoid marrow in femur, old penetration of skull and brain cortex by foreign body (knife blade).

Microscopical examination, by Dr. James Ewing:

Heart muscle shows considerable fatty degeneration of muscle cells.

Liver.—The liver cords are rather narrow and the capillaries contain an unusual number of cells. Many of these are large mononuclear leucocytes, but a considerable proportion are lymphocytes and nucleated red blood cells. The liver cells show marked granular degeneration everywhere, and in many foci there is intense acute granular hydropic and fatty degeneration, almost reaching necrosis. The deposits of pigment are moderate.

Spleen.—The Malpighian bodies are atrophic and the pulp sinuses and cords are difficult to identify on account of œdema and granular detritus. The cells of the pulp are deficient in number. There are many macrophages inclosing red cells and pigment, and many nucleated red cells mostly of large size.

Kidneys.—Cortical tubules are moderately dilated and tubule cells eroded. The glomerul appear normal. There is a slight diffuse growth of cellular connective tissue.

Stomach.—The mucosa shows a slight grade of chronic interstitial inflammation. New strands of connective tissue appear in many places and the relations of the glands are somewhat disturbed. There is no distinct atrophy of the mucosa.

The muscular coat shows slight interstitial myositis.

Marrow.—In the mid-femoral region the marrow is entirely fatty. Near the epiphysis there are patches in which the fat cells are surrounded by cords of large mononuclear cells, lymphocytes and nucleated red blood cells. In the ribs a few fat cells still persist. The marrow cords here contain a large number of mononuclear cells, neutrophiles and eosinophiles. The nucleated red cells are not extremely abundant and most of them are of large size, falling in the class of megaloblasts. There are no definite islands of nucleated red cells.

The following additional report of the miscroscopic examination of the central nervous system has been kindly supplied by Dr. James R. Hunt, who made the research:

Brain.—On removing the calvarium the end of a knife blade was found penetrating the skull and dura mater on the left side of the vertex.

Corresponding to the point of the blade, there was a rounded depression in the brain cortex, one-quarter of an inch deep, with a rounded margin, quite denuded of the pia mater. The brain substance surrounding the depression has a distinct rusty tinge.

The depression is on a level with the arm centre, but situated in the supra-marginal gyrus behind the posterior central convolution.

Microscopically this area was found sclerosed with numerous deposits of pigment, intra-cellular and surrounding the blood vessels.

The blood vessels were thickened and the cortical cells atrophied and sclerosed.

Otherwise the microscopic examination of the brain was negative. The vessels of Willis normal.

Spinal Cord.—Extensive degenerations are present in the posterior and lateral columns of the spinal cord, involving chiefly the columns of Goll, the crossed pyramidal tracts and the direct cerebellar tracts.

In these areas the sclerosis is advanced and but very little of the normal structure remains.

The field resembles the usual systemic degeneration, except that the replacement sclerosis is incomplete, producing an appearance of vacuolation.

The degenerations in the columns of Goll begin in the mid-lumbar region and increase in intensity upwards.

The direct cerebellar tracts are degenerated throughout their whole extent as are the crossed pyramidal tracts.

The latter can be traced to the sacral region.

The degenerations in the columns of Burdach while diffusely disseminated is not so compact as in the columns of Goll and is more of the vacuolation type, with falling out of minute areas of nerve substance.

Smaller areas of degeneration are scattered over the antero-lateral columns in the areas occupied by the direct pyramidal tracts, Gower's tract and the antero-lateral ground bundles.

These are in no sense systemic in character and present the usual vacuolations with absence and degeneration of the nerve fibres.

In the degenerated areas the whole nerve fibre may have been destroyed leaving a space: frequently, however, the axis cylinder is preserved, often normal, occasionally diminished in size.

The myelin sheath is not infrequently degenerated and undergoing fragmentation.

Numerous granule cells are scattered over the degenerated areas, some few containing myelin droplets staining black by the Marchi method. A few myelin droplets are also present in the perivascular sheath of the blood vessels, but the sections are singularly free from evidences of recent degeneration as indicated by the Marchi method.

The blood vessels, both of the gray and white matter of the cord, are thickened and sclerosed, especially evident within the diseased areas.

The lepto-meninges are not appreciably thickened.

The cells of the anterior horns show some chromatolysis and an occasional dislocation of the nucleus, but are otherwise normal.

It is interesting to note that all the nine foregoing cases were observed in adults between 37 and 66 years of age. Syphilis, alcoholism, privation, errors in diet and poor hygienic surroundings, although recorded in several cases, did not appear to be contributive factors with uniformity. In several cases arteriosclerosis was marked. The cases presented a uniform clinical picture, in which the important features were as follows: A lemon-yellow hue of the skin, with sometimes a distinct subicteroid color; great muscular weakness; dyspnœa, only on exertion; a low grade of irregular fever (100 degrees to 101 degrees F.); moderate œdema of the extremities; a systolic soft blowing murmur heard either at the base or apex of the heart, or both; a quick pulse (90–120), usually soft; headache; faintness on exertion; gastric symptoms, such as pain and dyspepsia, which were observed in some degree in all cases, although in only six was persistent vomiting recorded. There was a marked tendency to pulmonary congestion and œdema. In three cases enlargement of the lymph glands in various parts of the body was observed. In four cases nervous symptoms were absent (excepting variations in the knee jerks), but in three they were present in marked degree and variety. In only two cases was emaciation conspicuous. In no case was hemorrhage observed clinically, although ecchymoses were usually present at autopsy in the mucous membranes of the alimentary tract. The blood, in all those cases in which an exhaustive examination was made, showed fairly constant and typical changes, although the examinations illustrate the importance of their repetition, for in some specimens, for example, megaloblasts may be absent in the midst of a series in which they were present both earlier and later. The important characteristics of the blood in the several cases were: Marked oligocythemia and oligochromemia, with a normal, or frequently above normal, color index; marked macrocytosis, poikilocytosis and polychromatophilia; the presence of megaloblasts and gigantoblasts; a relative increase in percentage of lymphocytes, and often a normal number of white cells or else leucopenia.

Cases 8 and 9 illustrate extremely well the possibility of spontaneous improvement. I cannot attribute this fairly to any special treatment, for in Case 9, at least, the most marked improvement followed the temporary suspension of all treatment. It hardly seems possible that such improvement should occur when the hæmoglobin had fallen as low as 10 per cent. and the red cells to 550,000, as it did in Case 9, the count rising again to 40 per cent. and 1,667,000 within three weeks. Yet during this period, when the red cells numbered only one-tenth the normal, there was neither subjective nor objective dyspnœa, as the patient lay in bed. It was a great surprise to me that any of his functions could be performed at all, although I have twice witnessed a similar condition. In my former service at the New York Hospital, among the pernicious anæmias were two of which I have records, in which the blood examination showed respectively 539,000 and 508,000 red cells, and in the latter, three days before death, the red cells reached the almost incredibly low record of 217,500. As these cases were observed some fifteen years ago, before the development of modern methods of blood staining and accurate differential counts, I am merely able to refer to the gross report. Both patients died suddenly of asthenia, as might have been expected, and I recall the same absence of dyspnœa, of unconsciousness, of pulmonary œdema or any of the special symptoms that might be expected to arise from such impoverished blood, and death appeared to be due to the final exhaustion of the highly anæmic heart muscle.

The blood examination alone should not be relied upon too strongly for prognosis, for, as illustrated by this series of cases, death may take place when the red cells are above 1,000,000, or temporary improvement is possible when they number scarcely half that amount. More reliance for prognosis is therefore to be placed upon the patient's general condition.

Case 10.—The following very complete microscopic examination of viscera from a case of pernicious anæmia was from a Coroner's case at the hospital. Unfortunately no clinical data were obtainable, and a record of the gross pathology is also lacking. Being a Coroner's case, the patient probably died within a few hours after admission, and was possibly moribund, hence the absence of clinical history.

Microscopical examination of viscera.

Heart Muscle.—Shows the usual fatty degeneration and increase of pigment.

Lungs.—Oedematous.

Liver.—There is advanced fatty degeneration of long standing and a more recent and very active fatty and granular degeneration. The only fairly preserved cells are confined to a narrow zone along the portal canals. In the centres of most lobules all the cells are replaced by large fat globules. In the intermediate zone the cells contain smaller fat globules, granular and red-cells detritus, and pigment. Many are fragmented and the cords of cells are indistinguishable. There are a few small hemorrhages.

Kidney.—There is a diffuse growth of old acellular connective tissue throughout the cortex and most marked in numerous wedge-shaped areas in which most of the glomeruli are completely fibrosed. The remaining glomeruli are intact. The tubules have not suffered much except in the wedge-shaped fibrous areas where they are atrophic. The tubule cells show granular degeneration.

Spleen.—The organ is the seat of extensive growth of diffuse connective tissue. The pulp cords are obliterated, the cells deficient and the Malpighian bodies are very small. There are many phagocytes inclosing red cells and their detritus.

Marrow.—The bone-marrow is of the atrophic gelatinous type in the gross. Sections from a rib show persistence of a normal number of fat cells. The marrow cords contain an excessive number of red blood cells and the sinuses are congested and often much distended. In the cords, lymphocytes, neutrophile myelocytes, and polynuclear leucocytes are deficient. Eosinophile cells are much more numerous than is normal. There are no islands of nucleated red cells, but an excessive number of nucleated red cells, mostly megaloblasts, are scattered diffusely through cords and sinuses. There are a great many macrophages inclosing red cells and pigment.

Stomach.—Shows an advanced stage of atrophic gastritis. The muscular coat is thin. The mucosa shows diffuse growth of new connective tissue, atrophy of lymphoid cells and tissue, while the glands are very deficient in number and depth. In the intestinal mucosa the follicles of Lieberkühn appear to be deficient in number.

Pathological Diagnosis.—Atrophic gastritis, pernicious anæmia, megaloblastic degeneration of bone-marrow, fatty heart, acute and chronic fatty degeneration of liver, chronic diffuse nephritis, chronic interstitial splenitis.

The two autopsy records which follow are unaccompanied by clinical histories, for the reason that, owing to the patient's unconsciousness or moribund condition, none were obtainable.

Case 11.—*Autopsy*—Male adult. Skin of muddy yellow color, sclerotics yellow; muscles dark red. There was a half pint of bile-colored serum in both pleural cavities and about a drachm in the pericardial sac. There was evidence of a fibrous pleurisy, old adhesions, and a healed calcareous tuberculous cavity. The lungs were both anæmic and œdematous and the bronchi were filled with yellow frothy mucus. The papillary muscles of the heart showed fatty degeneration in patches, and there were patches of atheroma in the aorta. The mitral valve was thickened. The spleen was slightly enlarged and extremely soft, or pultaceous; its capsule was thickened. The liver was pigmented, but otherwise seemed normal. The kidneys were of the granular contracted type, and the apex of one papilla of the right kidney was the site of hemorrhage. The stomach mucosa was ecchymotic, but the organ appeared normal in size. The mucosa of the duodenum was œdematous and bile-stained. The solitary follicles of the ileum were markedly hypertrophied, hard and prominently elevated. The entire lumen of the intestine contained clots of decomposing blood. The brain was very anæmic and the pia mater œdematous. The total quantity of blood in the cadaver was remarkably small. The marrow in the femoral shafts was very red.

Case 12.—*Autopsy*—Male adult. The body showed extreme pallor of the skin and œdema of the feet and legs. In the chest there was œdema of the areolar tissue, especially in the mediastinum. About a pint of clear serum was discovered in the pleural cavities, one-half pint in the pericardium and a quart in the peritoneal cavity. The lungs showed evidence of extreme pallor and œdema, and at the apices there were old pleuritic adhesions. All the cavities of the heart were thin-walled and considerably dilated. The papillary muscles were hypertrophied, and the mitral cusps were thickened and beset with large marginal granulations. The aortic cusps were rigid and infiltrated with lime salts. The ascending aorta and coronary arteries were highly atheromatous. The spleen was slightly enlarged, the capsule adherent, and the Malpighian bodies were prominent. The liver was cirrhotic, of "nutmeg" type, moderately enlarged and with areas of perihepatitis. The kidneys exhibited a passive hyperæmia and parenchymatous degeneration. The connective tissue of the pancreas was increased. The stomach was small and atrophic; the pylorus was 1 cm. in diameter. The intestines were in a condition of chronic passive hyperæmia. The brain and cord were not examined.

The pathological findings in the six autopsies were remarkably uniform. In every case there was pulmonary œdema, fatty degeneration of the heart, and in five cases hyperplasia and other changes in the red bone marrow, and a chronic splenitis. In four cases the spleen was slightly enlarged, in two it was normal in size. The pancreas was twice found with fatty degeneration and once with interstitial pancreatitis. The liver was fatty in four cases, pigmented in one and cirrhotic in one. In two cases there was perihepatitis. Ecchymoses were observed in the gastro-intestinal mucosa three times and once in the pia mater. The latter was twice found œdematous. In one case the Peyer's patches were atrophic and in one hypertrophic. In five cases there was an advanced degree of chronic atrophic or interstitial gastritis, affecting chiefly the fundus and to a slight degree only the pylorus, which, however, was usually much narrowed. The muscularis was thickened in several cases, but in one it was thinned, and in several instances the mucosa was entirely gone in large areas, or the tubules were greatly distorted. Unfortunately, no examinations of the spinal cords were recorded except in Case 9.

Although the pathological findings in all these cases are very similar, it is not clear to what extent a *post hoc propter hoc* argument may be applied to the etiology. The disease is very chronic, often lasting several years, during which time the various fatty degenerations of the liver, pancreas and heart might easily develop as a result of changes dependent primarily upon malnutrition and deficient oxidation. The changes in the bone marrow and spleen, which have long been recognized in pernicious anæmia, and which were well illustrated by the autopsy cases of this series, correspond perfectly with the blood changes, and fit into the general picture of a distinct blood disease. The marked atrophic changes in the gastric mucosa (the chronic interstitial gastritis) present a more difficult problem in relation to the etiology. They are hardly such changes as would be expected to arise from anæmia alone, like the fatty degenerations of the other viscera, yet they are surprisingly frequent in cases of pernicious anæmia. It must be admitted that in not all cases does the stomach present the lesions of atrophic gastritis; and, on the other hand, complete abolition of gastric function—even total extirpation of the organ—is not followed by any such extraordinary degree of secondary anæmia as is represented by the typical "pernicious" type of primary anæmia. Again, extreme anæmia, very closely resembling the "pernicious" form in characteristics, may be secondary to definite diseases not involving the stomach, and may be completely recovered from, as illustrated by Cases 13 and 14 of this series. Such recovery could hardly take place if permanent gastric atrophy, with loss of tissue, were a *sine qua non* of extreme anæmia. It should be observed, however, that the gastric mucosa may appear normal macroscopically, and yet the microscope may reveal chronic interstitial inflammation, as illustrated by the records of Case 9.

Case 13 was that of an Irish laborer, 33 years old. He was very alcoholic, but gave no venereal history. Two months prior to admission to the hospital he complained of swelling of the feet and legs, headache, vertigo, dyspnœa, weakness and numbness of hands and feet with epigastric pain. These symptoms began gradually, compelling him to quit work after a month. On admission, the patient appeared fairly nourished, but of a very pale yellow color. The heart was slightly enlarged, and there were systolic and presystolic mitral and systolic aortic murmurs. The arteries appeared normal, as did all the viscera except the heart. The reflexes were normal; the epitrochlear and inguinal glands were enlarged; dyspnœa and orthopnœa were pronounced. Examination of the stomach showed diminished motility and anacidity. For six weeks the patient had a low grade of intermittent irregular fever in which the temperature occasionally rose to 101 degrees F. The pulse ranged between 80 and 100. The urine contained a trace of albumin with hyaline and granular casts.

The stools were examined for parasites with negative result. On admission, the patient's blood was that of pernicious anæmia, with moderate poikilocytosis and polychromatophilia, and in all the earlier examinations there were many large forms of cells with basophilia and anisocytosis. Under treatment with diet, rest, iron, arsenic and fresh air, the patient recovered completely after four months from all symptoms except the heart murmurs, the chronic catarrhal gastritis improved and the red blood cells became normal. The blood analyses are stated in the following table:

	Sept. 15, 1902.	Oct. 9, 1902.	Oct. 10, 1902.	Oct. 11, 1902.	Oct. 22, 1902.	Nov. 1, 1902.	Nov. 11, 1902.	Dec. 1. 1902.
Hæmoglobin	23	15	15	20	48	80	80	80
Red cells	1,036,000	1,100,000	927,200	1,248,000	2,152,000	3,072,000	3,584,000	4,096,000
White cells	4,000	4,000	4,000	3,000	2,000	2,000	3,000	8,000
Color index	1.1	0.7	0.8	0.8	1.1	1.3	1.1	0.98
Normoblasts	2	0	5	4	0	0	0	0
Polynuclear	60. per cent.	54	55.6	55.6	42	32.3	66.3	66.75
Lymphocytes	28.8 " "	33.	37.	27.6	20.6	33.6	33.	13.25
Large mononuclear	8.6 " "	11.6	5.9	11.3	28.1	24.6	25	18.75
Eosinophiles	0 4 " "	2.4	1	1.3	5	8.2	0.3	1.
Mast cells	0.2	0.5	0	0.3	1	1.3	2	0.25

Case 14 was that of a Finn, 17 years old, employed as a plumber's boy. The patient gave no family history of importance or any personal history until about seven months prior to admission, when he first noticed increasing pallor. A month later he had repeated attacks of epistaxis on successive days, one of which lasted half a day. He also began to suffer from dyspnœa on exertion. A month before admission his legs became œdematous, and as his weakness increased and he lost some 30 pounds in weight, and had had about fifty attacks of epistaxis, he applied to the hospital for relief. A month before admission, however, he took a dose of a decoction of some seeds which had been sent him from Finland to rid him of a tapeworm of several years' growth. He passed four feet of the worm, as he thought, with the head attached.

Upon admission, the boy presented a typical clinical picture of pernicious anæmia. He appeared well nourished, but was of a pale lemon yellow color. There was moderate œdema of the legs, and the cervical glands were enlarged upon the left side. The pulse was rapid and compressible; the heart appeared normal in size, but presented an apical systolic bruit, and another at the base, transmitted to the vessels of the neck upon the right side; the pulmonic second sound was accentuated; the leg reflexes were diminished, but there were no other nerve symptoms; the abdominal viscera appeared normal; the urine was slightly albuminous, with a few hyaline casts, and uniformly low specific gravity, which on repeated examination was between 1007 and 1010; there was moderate dyspnœa, and a few crepitant râles were heard at the base of the right chest. The patient had repeated epistaxis, but examination of the nasal cavities revealed nothing abnormal. Dr. Alexander Duane, who examined the eyes for me, reported the optic discs as very pale, with small veins and arteries, secondary atrophy, and several small hemorrhages in the fundus, but none in the maculæ or periphery; motion of the eyes and vision were normal, but the patient had had a transient attack of blindness in the left eye the week before the examination. The patient had several severe attacks of epistaxis, which were controlled with difficulty. The temperature on admission was 105 degrees F. for three days with a pulse of 120, and for two weeks thereafter it ranged between 100 degrees and 101 degrees F., becoming subsequently normal. Soon after admission the ova of the *Bothriocephalus latus* were observed in the feces, with a few red blood cells. During a period of eight weeks the patient was given five doses of the oleo. resin of aspidium, 1 ounce in each dose, and on four occasions thereafter he passed several feet of the parasite—in all about 25 feet—but no head was discovered. The dose of male fern was preceded by starvation and followed in 12 hours by castor oil. In addition, the patient received adrenalin chloride, Fowler's solution, Basham's mixture and sodium cacodylate. At the height of his symptoms he had incontinence of both urine and feces. After three weeks of treatment the hemorrhages ceased, and the general condition, including the anæmia, began to show steady improvement, which was maintained until the patient was discharged, after three months, apparently cured and with normal blood. A table of six representative blood analyses follows:

No.	DATE.	R. B. C.	W.B C.	Hæmoglobin.	Color Index.	Polynuclear.	Lymphocytes.	Largemononuclear.	Tranitional.	Eosinophiles.	Mast Cells.	Normoblasts.	Megaloblasts.	No. of Cells Counted.
	1904.													
1	Apr. 4	608,000	2,000	20%	1.6	70.1%	26.3%	2.9%	..	0.3%	0.3	26	0	600
2	" 7	760,000	3,600	20	..	60	24.5	11.5	4.%	0	0	3	4	200
3	" 16	1,000,000	3,300	20	1	65	22.5	6	4.5	2.%	0	6	2	400
4	May 13	2,884,000	6,000	60	1.19	77.5	10	4	3.5	4.5%	0.5	0	0	300
5	June 14	3,776,000	10,000	85	..	60	16	5	9	9.%	0	0	0	300
6	Nov. 24	5,980,000	10,000	98	..	55	26 6	6.3	5.6	5	0.6	0	0	300

In the first and second examinations there was decided anisocytosis, poikilocytosis and polychromatophilia with occasional basophiles, and only very few large red cells. In the third and fourth examinations these characteristics all became much less decided, normoblasts and megaloblasts disappeared, and in the last (sixth) examination there remained only slight basophilia, a very few small pale red cells, and the red cells presented very little variation from the normal in size or shape.

(This case was studied in the service of Dr. C. L. Dana and the writer.)

The last two cases (Nos. 13 and 14) present a very interesting condition, for in both the anæmia reached a degree which was, as one would suppose, incompatible with prolongation of life, yet both patients recovered from the anæmia. In Case 14 the anæmia was clearly secondary to toxæmia caused by the *Bothriocephalus latus* but was doubtless much intensified by the repeated hemorrhages that took place. In Case 13 the anæmia appeared to be secondary to chronic endocarditis and gastritis, yet it attained a degree far beyond that which is usual in such cases.

The following statistical summary is appended:

Of the 14 cases there were 9 males, 4 females, and 1 with sex unrecorded (microscopic examination only). Omitting the 3 cases in which there are only pathological records, leaves 11 cases. Of these 11 patients, 7 had fever, the temperature ranging from 100 degrees to 105 degrees F.; 7 had dyspnœa; 6, severe vomiting and other gastric symptoms; 5, enlarged lymphatic glands; 4, nervous symptoms (other than altered knee jerks); and 1 only had hemorrhages (the Bothriocephalus case, No. 14).

The ages of the patients in the two secondary cases were respectively 17 and 33 years. In all the idiopathic cases the ages were more advanced, being as follows: 66, 55, 52, 49, 49, 47, 44, 38, 37. The minimum hæmoglobin and red cell count reached in each case was as follows:

Hæmoglobin.	Red Blood Cells.
?	2,024,000
50 per cent.	1,872,000
40 "	1,488,000
25 "	1,084,000
20 "	1,008,000
22 "	1,000,000
15 "	927,000
10 "	856,000
18 "	848,000
20 "	608,000
10 "	550,000

RESULTS OF OPERATIVE TREATMENT FOR CERTAIN CASES OF ADVANCED MALIGNANT DISEASE.

By Benjamin T. Tilton, M. D.

When called upon to treat advanced cases of malignant disease, it would seem proper for the surgeon either to dissent entirely from any operative interference or employ only radical methods. To subject a patient to an incomplete operation, which manifestly cannot but result in recurrence, is unjustifiable. It disappoints the patient and often results in a condition worse than the original one. We must be in a position to go well wide of the growth or otherwise give up any idea of operative treatment beyond that connected with palliative measures.

Radical measures may imply, however, the sacrifice of considerable tissue, the loss of nerve supply to the part and marked disfigurement. In order to expose sufficiently the field of operation and to follow with the eye the limits of the disease, it is often necessary to make extensive resections of bone and lay open important cavities. Deep dissections of lymph glands are frequently required. In order to cut off the blood supply temporarily or to starve the region permanently, the ligation of one or several large arteries will often have to be resorted to. The defects resulting from the sacrifice of a large area may later require extensive skin grafting.

In the cases that follow radical measures were imperative on account of the advanced stage of the disease, and every means was used to eradicate the entire growth and prevent recurrence. In all of the cases the chances of complete cure were not bright, but in none was the outlook hopeless. Wide excision of the growth was employed, when possible the blood supply of the part was cut off by ligation of the main artery, the thermo-cautery was used in suspicious places after excision, and lymph glands in the vicinity were always excised. The success of the operative work would seem to be due to the combination of these measures.

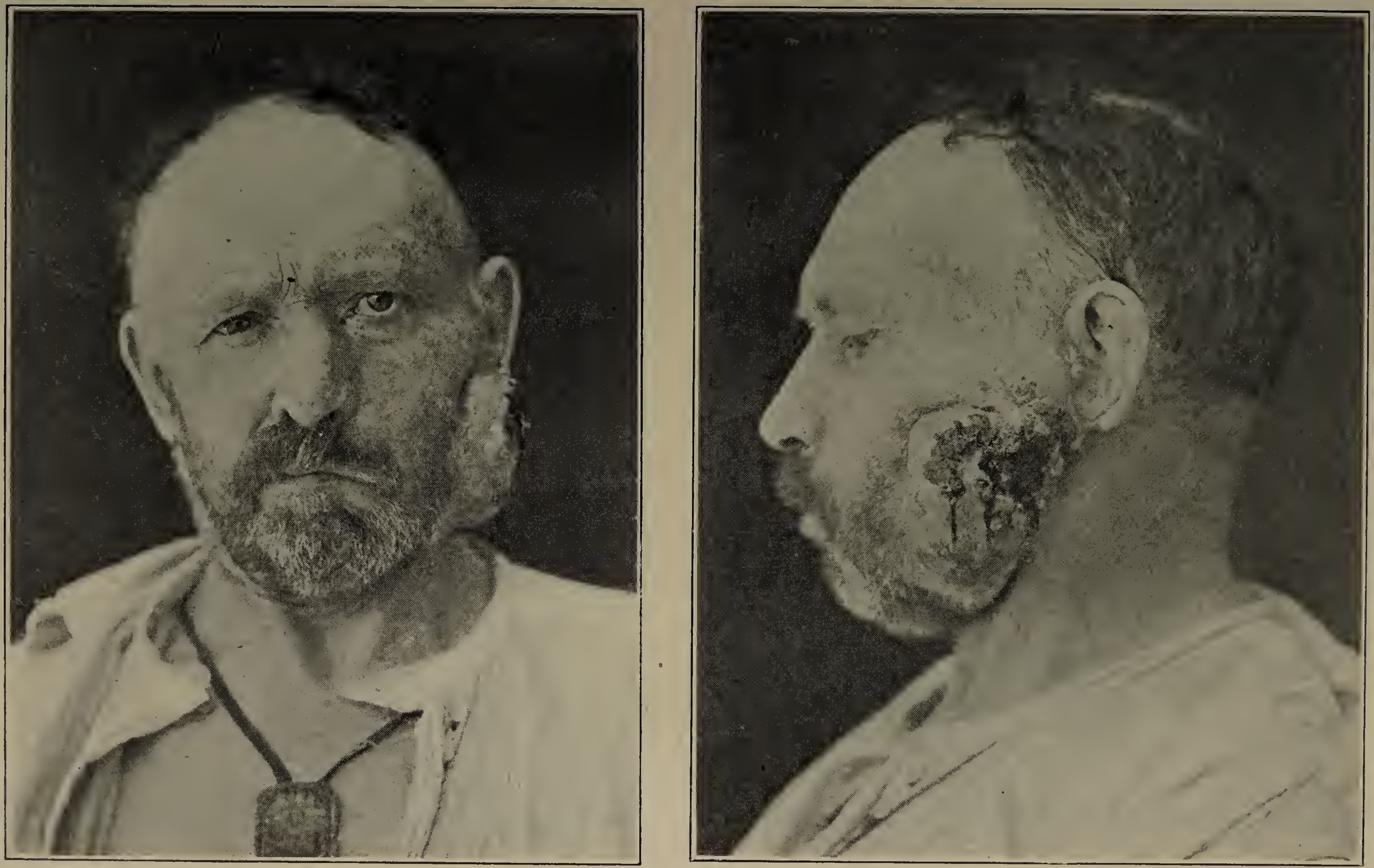
I should like to lay special stress upon the use of the thermocautery in connection with excision. It seems to be useful in preventing recurrence, and I feel that it ought to be used in a more routine way in these desperate cases. Ligation of the main artery facilitates dissection and no doubt helps in preventing recurrence.

CASE I.—*Recurrent carcinoma of the face; ligation of both external carotids; excision; skin grafting.*

This patient, a laborer, 53 years of age, was admitted to Bellevue Hospital September 22, 1903. His family history was negative. Up to two years ago he had always been well. At this time he noticed a small nodule in the region of the angle of the jaw; it grew rapidly and soon reached the size of a large egg. On February 2, 1903, he entered Bellevue Hospital. At this time the growth appeared to the surgeon on duty to be inoperable and he was transferred to the Metropolitan Hospital, on Blackwell's Island. He was operated upon there a few days later, the growth being excised and the facial nerve divided, causing facial paralysis. He was discharged from the hospital in July with a recurrence at the site of operation the size of a walnut.

On September 22, 1903, he entered Bellevue Hospital for the second time. The secondary growth had now begun to ulcerate and bleed freely. The patient was losing flesh and strength. He complained of great pain in the region of the growth, extending up to the head and sought eagerly for relief. The condition is shown in the accompanying photograph taken at this time.

Operation September 29. The two external carotid arteries were tied, an incision being made along the inner border of the sterno-mastoid muscle, and a few hard lymph glands were removed from the left side. The tumor was now excised by a curved incision circumscribing the growth and going well wide of its limits. It extended forward, about three fingers' breadth, from the ear downward below the ramus of the jaw and backward to a point over the mastoid process. A part of the lower portion of the left ear was sacrificed. There was very little



Case I.—Recurrent Carcinoma of Face. Ligation of both External Carotids; Excision; Skin-grafting.

bleeding. This extensive area was cauterized in places with the thermo-cautery and covered with sterile gauze dressing which exerted pressure. The operation was followed by considerable shock from which the patient, however, rallied in a few days.

Second operation November 5. The area of granulations had diminished very markedly in size. Skin grafts were taken from the thigh and placed upon the granulating surface which was previously scraped with a sharp spoon. The skin grafts took satisfactorily and in six weeks the wound had healed solid.

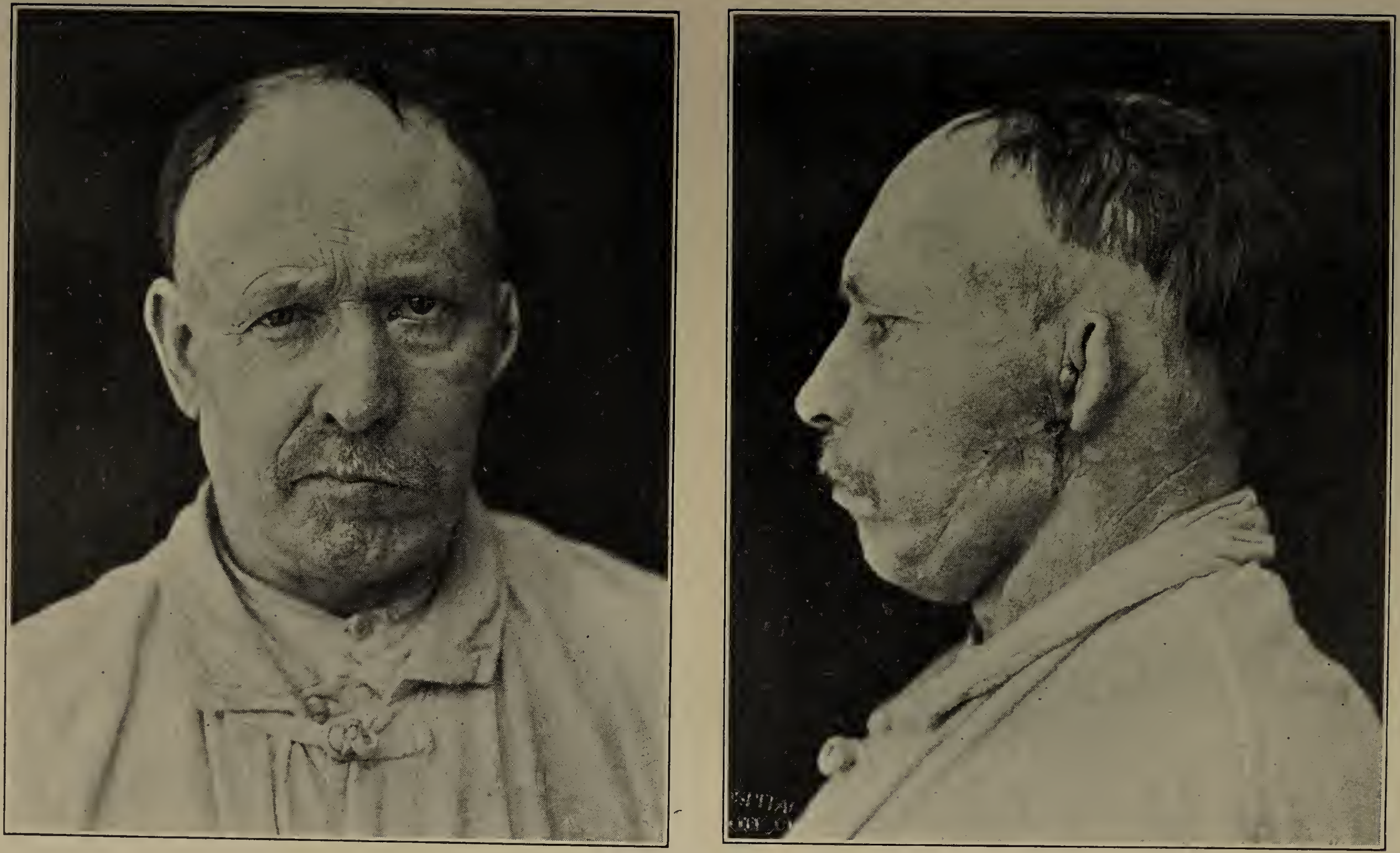
The patient has been seen several times since the operation and is now doing his work. Although seven months have elapsed since the operation there are no signs of recurrence of the growth. He has gained several pounds in weight and seems perfectly well. The cure is a satisfactory one owing to the apparent great malignancy of the tumor which caused its rapid recurrence after the first operation. The ligation of the two external carotid arteries made the excision bloodless and easy.

Case II.—*Epithelioma of the tongue, with extensive glands of the neck.*

This patient, 55 years of age, was admitted to Bellevue Hospital March, 1904. He complained of a tumor of the tongue, which he had noticed the past nine months.

Examination showed a hard, ulcerating tumor of the left border of the tongue extending back to the epiglottis and involving the floor of the mouth on that side. There was a swelling on the left side of the neck consisting of enlarged and indurated glands. The diagnosis of epithelioma was made by examination of a small section taken from the edge of the tumor. The patient was losing flesh and strength rapidly.

Operation March 15. A curved incision was made over the swelling in the neck from the middle of the jaw downward and inward toward the thyroid cartilage. Through this incision several carcinomatous glands were removed and the external carotid artery tied. The wound cavity was then packed with gauze. An incision was now made from the left angle of the mouth outward to the masseter muscle, then downward along



Case I.—Result of Operation.

the anterior border of the muscle to the lower border of the jaw, where it met the first incision. The lower jaw was then sawn through and the cavity of the mouth and pharynx thus thoroughly exposed. There was no difficulty in excising the entire tongue, the floor of the mouth on the left side, and the anterior pillar of the fauces. The exposure made accurate dissection very easy. The hemorrhage was slight and easily controlled.

The large wound cavity made by the removal of the tongue and other structures was drained with gauze which was brought out through the lower angle of the primary incision. The bone was sutured with kangaroo tendon. The patient made a good recovery from the operation. He is still in the hospital with a slowly healing sinus in the neck, but there seems but little probability of a local recurrence. There are no glandular enlargements in the neck. The patient's general condition is improving rapidly and he is gaining weight.

CASE III.—*Carcinoma of the penis, with secondary growths in both groins.*

This patient, 40 years of age, was admitted to Bellevue Hospital March 17, 1901.

Examination showed a large ulcerating growth on the under surface of the glans penis. The tumor was of one year's duration. Two months before his admission he had been circumcised by his family physician. The patient was losing flesh and strength rapidly. There were enlarged glands in both groins.

Operation March 20, 1901. The penis was amputated close to the symphysis and the mucous membrane of the urethra sewn circularly to the skin incision. The inguinal region was exposed on both sides and several hard glands were removed by careful dissection.

The patient made an excellent recovery. Three years have elapsed since operation. He is able to urinate without the employment of artificial means and shows no signs of recurrence. He has gained twenty-five pounds in weight and is able to perform his duties as a carpenter.

Case IV.—*Carcinoma of the breast, with extensive involvements of the skin and axillary glands.*

This patient, 65 years of age, was admitted to Bellevue Hospital in August, 1902. She complained of a swelling of the breast which had lasted about a year.

Examination showed an ulcerating scirrhus of the left breast with distinctly enlarged glands in the axilla. There were small, hard nodules in the skin for a considerable distance about the growth and the latter was adherent to the chest wall. The patient was losing flesh and strength rapidly and had become considerably emaciated. Owing to the extensive involvement of the skin a complete cure seemed doubtful.

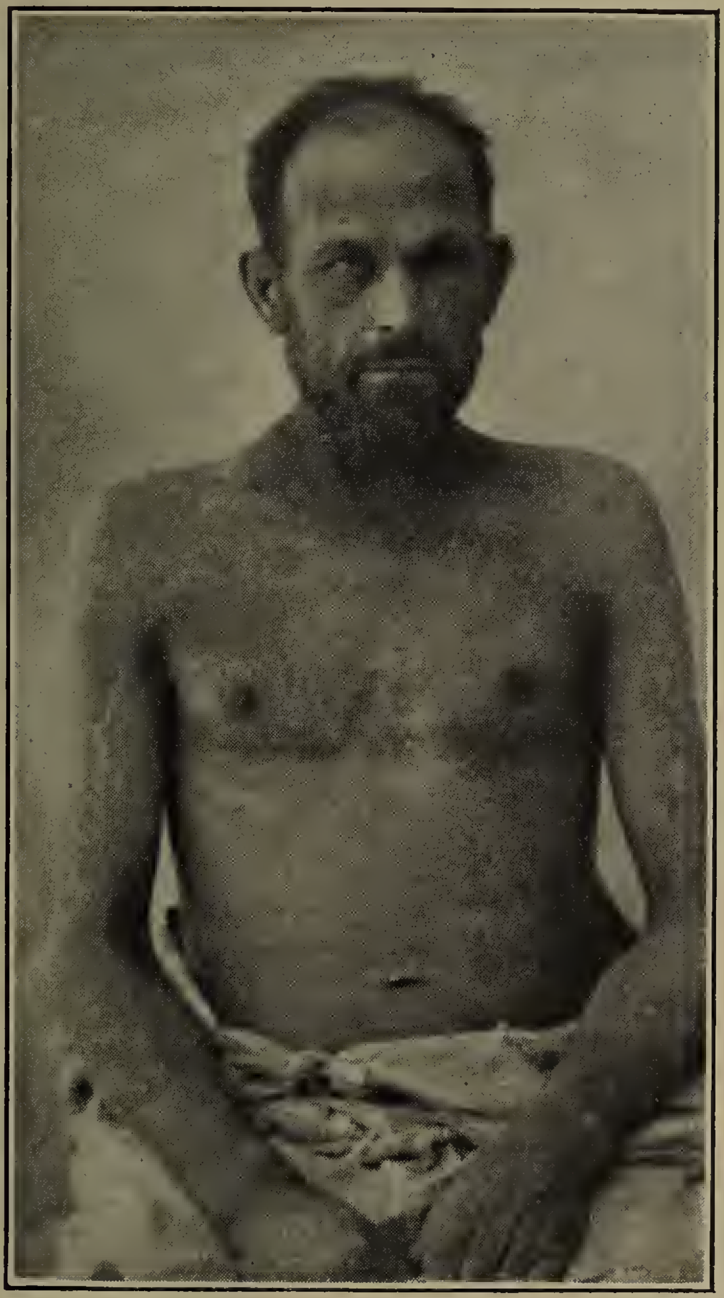
Operation August 16, 1902. The Halstead incision was employed, through which most of the pectoralis major and the whole of the pectoralis minor muscles were removed with the breast and the tumor. The periosteum of the ribs was removed with the periosteal elevator. The skin incision went very wide of the tumor and cutaneous nodules. The axilla was opened and the enlarged glands removed up to the apex. It was impossible to bring together the skin margins owing to the large amount of tissue removed, and consequently the extensive wound was allowed to granulate. Eight weeks later the granulating surface was covered with skin grafts taken from the thigh.

At the present time, one year and eight months after operation, the patient is in good health. She feels much stronger than before the operation and has gained considerably in weight. Her appetite is good and she shows no signs of cachexia. There are a few small nodules scattered in the skin in the region of the cicatrix. These are not painful and show no tendency to grow rapidly or extend. There is no sign of recurrence in the cicatrix or axilla.

TREATMENT OF HODGKINS' DISEASE BY MEANS OF X-RAY BATH.

By Egbert Le Fevre and Samuel A. Brown.

In the *Journal of Medical Research,* June, 1903, Dr. Channing Simmons reports the pathological findings and hæmotology of nine cases of Hodgkins' disease. This was supplemented in the *Medical Record* of May 14, 1904, by the report of the ninth case of Dr. Simmons' series by Dr. E. Finch, who outlined in detail a plan of treatment with the X-ray and gave the results obtained in this particular case.



Hodgkins Disease.

In view of the extremely satisfactory results obtained by Dr. Finch it was considered advisable to use the same treatment in the following case:

Joseph Smith, age 39, painter by occupation, was admitted to Third Medical Division of Bellevue Hospital December 1, 1903, with the following history:

Family History.—Negative except that mother died of " stone cancer " of breast at age of 53. Wife living and well. Three children living and well.

Personal History.—Habits—Irregular in his use of alcohol; total abstainer for months, then again would take eight or ten whiskies for several days at a time. Four and a half ounces tobacco per week. Venereal—Gonorrhœa twenty years ago; good recovery; no sequellæ. Denies syphilis.

Past History.—Diseases of childhood, negative. Fourteen years ago patient states he contracted rheumatism in his right ankle, which began to swell, but did not get red. It did not affect any other joints. Believes he " caught cold " in it. Other diseases negative except " la grippe " three years ago. Patient was ill three or four weeks. Cough persisted about six weeks. Two and a half years ago patient again suffered from rheumatism. Various joints of body were swollen, but were not red. Patient was ill nine weeks. Was not able to lie in bed because of pain. Was compelled to sit up in a chair.

Present History.—One year ago last April, 1903, patient first noticed a large swelling in right side of neck which was larger at that time than it is at present. Patient does not know of any cause which brought this about. One year ago, December, 1902, patient exerted himself tremendously by lifting a heavy sleigh; three or four weeks later patient observed a large swelling on left side of neck; this is also smaller than it was ten months ago. At same time (one year ago) patient began to be troubled with shortness of breath and inability to take a deep breath, not because of pain, but simply because he could not. No pain at that time. Patient complained of general weakness at that time. Last February, 1903, patient became very weak and could not perform the least task without sweating profusely. Was ill in bed five weeks. Since last August patient occasionally has a sharp pain in region of heart which shoots down left arm as far as elbow.

Cough has been troublesome since August. Expectoration moderate in amount. No blood in sputum. Appetite has always been good. At times appetite is ravenous. No bowel symptoms. Patient states during certain times that he would not urinate for twelve hours; then again would be compelled to arise once or twice at night to urinate.

PHYSICAL EXAMINATION.

Patient well nourished. Temporal arteries visible and tortuous. Right pupil reacts to light and accommodation. Conjunctivæ and mucous membranes injected. Left eye absent, lost through injury. Numerous small scars over face and cheeks. Axillary glands much swollen. Cervical very much swollen, and form large tumor mass on either side of neck. Superficial veins of thorax somewhat dilated, especially over left side. Visible pulsations seen in neck. Numerous small capillary dilatations over lower zone of thorax. Many brownish spots seen over body, some lighter in color and covered with a fine white scale. Skin very dry, and over neck, hands and feet epidermis is thick and leathery. Respirations are rapid, wheezing, cough distinctly stridulous, voice husky at times.

Abdomen distended, tympanitic all over, except epigastrium and right hypochondrium, where numerous small nodules are felt in anterior abdominal wall. Inguinal glands enlarged. A reducible left inguinal hernia is seen.

Mucous membranes good color. Tongue broad, moist, thickly coated, pale at edges, coarsely tremulous.

Throat.—Pharyngeal mucous membrane, pale. Follicles prominent.

Pulse.—Rapid, irregular, forcible, large, lumen obliterated with some difficulty. Heart—Apex beat not visible or palpable. Heard with maximum of intensity in fifth intercostal space, 4½ inches to left of median line. Apical sounds are distant, but distinct. No murmurs heard. Basal sounds similar to apical.

Lungs.—Anteriorally—On right side: Slight dullness for about 1½ inches to right of sternum, extending down and becoming continuous, with cardiac dullness. Left side: Area of dullness begins just about 1½ inches within anterior axillary line. Extends inward and downward, becoming continuous, with deep cardiac dullness. Over this area vocal fremitus is absent. Bronchovesicular breathing and bronchophony are obtained. Over rest of right lung percussion normal, harsh respiratory murmur is heard. On left slight dullness and diminished respiratory murmur. A few fine moist rales are heard. Posteriorally —On right, dullness above and in interscapular regions on percussion. Palpation gives diminished fremitus above and increase below midscapular region. Breathing bronchovesicular over region of dullness and is exaggerated below. A few small moist rales are heard. On left posterior, dullness and slightly diminished fremitus, bronchial breathing, bronchophony heard down to midscapular region. Respiratory murmur, vesicular below this level. A few moist rales are heard.

Liver.—Dullness begins at upper border of fifth rib and extends downward into and becomes continuous with dullness in abdomen. Anterior border can be felt at free border of ribs in right hypochondriac region.

Spleen.—Percusses enlarged, but is not palpable.

CLINICAL DATA ON ADMISSION.

December 2, 1903.—*Urine:* Yellowish, straw; acid, 1,029; alb., 0; sug., 0; urea, grs. viii. to ℥; excess of amorphous urates.

December 3, 1903.—*Blood:* Reds, 3,860,000, 70 per cent.; hemoglobin, 10,200 (10 A. M.); whites, 22,800 (4 P. M.).

December 2, 1903.—*Sputum:* No tubercle bacilli found.

The most important features in this case at the time of admission to the hospital were the great enlargement of the subclavical, axillary, mediastinal, retroperitoneal and the inguinal glands, and the fact that the glands on the right side of the body were very much larger than the corresponding glands on the left side.

Photographs showing the enlargement of the superclavical glands, the distended superficial vessels and the rough skin, are appended.

The mediastinal mass was extremely large, and on percussion the right border extended to the junction of the middle and outer third of the clavicle. On the left the dullness was continuous with the cardiac area. The encroachment on the right side had almost completely obliterated the lumen of the right bronchus, so that a condition of atelectasis existed in the right lung. The heart was displaced downward and to the left, and the rhythm markedly disturbed.

The fluoroscope confirmed the facts determined by the physical examination.

As a result of the enlargement of the glands there was great interference with the return circulation. The superficial veins were distended and there was an accumulation of fluid in both pleura.

Patient stated that shortness of breath and other pressure signs had increased gradually and progressively. At the time of admission he was unable to assume a recumbent position. He was running a slightly irregular temperature of 99 to 101, pulse 96 to 106, respirations 36 to 40. Clinical data on admission given above. Patient was placed in bed with a bed rest and allowed to sleep in a semi-recumbent position or in a chair alongside of the bed.

December 1, 1903, at the time of admission, he was voiding on the average of forty ounces of urine per day. He was then placed on the following treatment: Fowler's solution, 5 mm.; White drink, half ounce three times a day, and saline purges were given in order to relieve, if possible, the accumulation of fluid. It was also necessary to give the patient morphine at night to rest.

The patient's symptoms continued to increase in severity, weakness and dyspnœa especially.

December 30, 1903.—Chest was aspirated and fifty ounces of straw-colored fluid removed from right side. As the treatment has not controlled the disease, the suggestions outlined by Dr. Finch at the meeting of the Bellevue Hospital Society were adopted, beginning January 7, and the patient's condition at this time was as follows: *Urine:* Average quantity voided, xxxii. ℥; amber; acid, 1,031; albumen, 0; urea, grs. 216, in 24 hrs.; spec., ℥ xxiv.; heavy sediment; great excess amorphous urates; no casts. *Blood:* Shows a decrease of reds from 3,860,000 to 2,980,000; hemoglobin increase, 10 per cent., and the whites diminished to 8,000.

Oedema of lower extremities marked and accumulation of fluid in chest increasing on both sides. There was no decrease in the size of the glands up to this time.

On January 7, 1904, patient, under the direction of the House Staff, was exposed to the X-ray for a period of forty minutes. The target of the tube was placed within six inches of the surface.

The first exposure was made over the superclavical glands on the right side, this exposure lasting ten minutes. The tube was then moved to left side over the corresponding glands and remained in this position ten minutes. From the left side it was moved to the median line on the level with the fourth rib, where it remained for a period of ten minutes. The fourth and last exposure was made over the lower part of the abdomen, also for a period of ten minutes, making a total of forty minutes for the first exposure.

Patient was placed on the following medication at this time: Three grains quinine, five grains caffeine, six ounces inf. Trit. Repens three times a day, with sufficient heroin to control the cough, previous medication being cut.

The X-ray treatment was continued daily. The period of exposure was increased five minutes each day, until patient received a treatment extending over a period of one hour and forty minutes, tube being moved every ten or fifteen minutes to another part of the body.

The repeated and long application of the X-ray failed to produce burning or dermatitis. This was attributed to the fact that a very high tube was utilized. Patient was kept under careful daily observation, and a marked improvement was noticed ten days after beginning of treatment. The glands decreased in size and patient's general condition constantly and progressively improved.

On January 30 physical examination showed that the area of enlargement to the right of the mediastinal glands had decreased two inches, the fluid had disappeared from pleura, the dyspnœa was greatly relieved, the patient was able to assume a recumbent position, and his general health was so much improved that he was able to assist in the light work of the ward. He was able to obtain from eight to ten hours' sleep, as compared to two or three hours in his previous condition.

The condition of the mediastinal glands was verified by means of the fluoroscope at this time. The neck had decreased in size from twenty-two inches to fourteen, and other glands were proportionately decreased in size.

Patient remained under observation until February 11. His improvement continued without interruption. He was able at this time to go up and down stairs easily.

A slight dermatitis developed at this time and it was considered advisable to discontinue the treatment. He was advised to go to the country, but during his absence he was to report weekly. He was discharged February 16.

Patient remained in the country until April 8, a period of about seven weeks. He was readmitted April 8 with the following history:

During the first week after leaving the hospital he contracted a slight cold, but other than this he was in extremely good health, and continued to gain in strength rapidly and increasing his weight eight pounds. He was able to perform a considerable amount of work on a farm, stating that he had in one day sawed and split a cord of wood. His skin became much smoother and many shades lighter in color.

This improvement continued until one week before his readmission. He then noticed that the glands were beginning to enlarge again, and he entered the hospital complaining of shortness of breath and swelling of abdomen.

He was re-examined on admission and the glands, with the exception of those in the neck, were found to be almost, but not quite, as large as when first observed. He was advised to begin X-ray treatment immediately.

All signs of dermatitis had subsided and patient states that the skin is smoother and lighter in color than it has been for over a year.

It would seem from the results obtained in this case that we have in the X-ray an almost specific treatment for control and the relief of symptoms occasioned by the general lymphoid enlargement of this distressing disease. We believe that in the type of Hodgkins' disease, when accompanied by large glands that do not coalesce, gives better results with this than any other form of treatment so far suggested. Without further observations we are unable to determine whether or not it would be possible to continue the X-ray treatment until the glands would permanently remain small, but we know that we can at least relieve the patient of the symptoms, and by periodic exposures of from one to two weeks to the X-ray prevent the re-enlargement of glands.

THE TUBERCULOSIS CLINIC OF THE BELLEVUE HOSPITAL OUT-PATIENT DEPARTMENT.

James Alexander Miller, A. M., M. D.,

This clinic was first opened in December, 1903. It is held every afternoon, excepting Sundays and holidays, from 1 P. M. to 4 P. M., and is in the same building but in an entirely separate room from the other classes of the Out-patient Department. In connection with the clinic there is also a tent pavilion upon the hospital grounds, which accommodates twelve patients.

The whole clinic is under the general supervision of one director, who is also attending physician to the tent patients, but it is separated into four classes corresponding to the four medical divisions of Bellevue Hospital, and the patients of each class are under the immediate charge of an attending physician who represents his division and has the same duties and privileges as the attending physicians of the other clinics. These four attending physicians to the clinics are also assistant attending physicians to the tent patients.

As a general rule all patients are first referred to the clinic in general medicine and are transferred to the tuberculosis clinic after a diagnosis of tuberculosis has been made. If, however, a patient is specifically referred to the tuberculosis clinic by any outside physician or institution, he is admitted directly to that clinic, and printed cards to facilitate such reference of patients have been provided.

The main objects attempted in the institution of this clinic were:

1. *Careful and thorough medical attention to* each patient in a separate clinic by physicians especially interested and experienced in this disease.

2. *Systematic investigation and supervision of the home conditions* of each patient.

3. *Education of the patients and their families* in the principles of good hygiene and the prevention of the spread of infection.

4. *The study of the social and economic conditions* in connection with the medical aspects of each case and the relief and amelioration of those which are unfavorable.

5. *The opportunity for scientific investigation* and clinical experience for physicians and students.

Let us consider each one of these objects and note how we have attempted to attain them.

1. *Careful medical attention.*—When a patient is referred to this clinic he is sent to a separate room for tuberculous patients, the temperature and pulse are noted by the nurse in attendance, and the history is taken by one of the physicians upon special forms upon which all the points to be investigated are printed. A careful examination is then made, including chest measurements, laryngeal examination, if at all indicated, and careful weighing with the patients stripped to the waist. The results of this examination are recorded on the history sheet, the physical signs by the graphic method. A positive, or even probable diagnosis being made, the name and address is given to the nurse as a case to be visited. The sputum is either obtained immediately or a bottle given for its collection before the next visit, the specimen being sent to the Department of Health for examination. The patient is informed of the nature of his disease, of the principles of its treatment, and the dangers of infection, and printed circulars of instruction are given him for reference. The general principles of fresh air, rest, and extra nourishment, are explained in detail. As a general rule the diet is advised to be a general mixed one, with two quarts of milk and four raw eggs additional each day. This number is gradually increased to the point of increase in weight or the tolerance of the individual, but the maximum quantity prescribed is about three quarts of milk and eight to ten eggs daily. We find very few patients who can exceed this amount with any advantage, for it is undesirable to take away an appetite for mixed food and disastrous to overload the stomach beyond its power of digestion. The amount of fresh air and rest that these patients can obtain is, of course, very variable. It is almost always less than is desirable, but by

persistent emphasis upon their importance much can often be accomplished.

Medication plays a very minor role in our plan of management. With this comparatively ignorant class of patients some medicine must often be given simply for the moral effect. Drugs so given are, of course, simple and, at any rate, harmless. Cathartics are most necessary during overfeeding, and stomachic tonics are very useful. Cod liver oil is given freely as a food during the winter months. Creosote is very seldom prescribed. Symptoms such as distressing cough, pain, night sweats, etc., must be relieved. Our general principle, however, is to use as little medicine as possible. Pocket sputum pouches, and also more substantial ones for home use, are given to each patient from the drug room. All medicines and cuspidors are dispensed without charge. Patients are requested to return to the clinic once a week, and a record of the weight, temperature, pulse and general condition is made at each visit. Careful physical examination is made at least once a month, but, of course, as much oftener as any indication suggests.

2. *Thorough investigation and supervision of the home conditions* of each patient. This is best accomplished by means of visiting nurses who have had special training in district nursing as well as hospital work. At Bellevue we have two such nurses, Miss Damer and Miss Hopkins. Both are graduates of the Bellevue Training School, and both have been active and successful in district work; the former with the Buffalo Charity Organization Society, the latter with the Charity Organization Society of New York. Under their direction one of the pupil nurses also helps in this work, which thus becomes a part of her training.

Each new case is referred to the nurse with any especial instructions or information that may be thought advisable, and, after a thorough inspection of the home, a report is made out by her upon printed forms provided for the purpose and submitted to the physician in charge of the case, together with any suggestions which may occur to her as a result of her visit.

Our general plan is to arrange the whole domestic economy to the best interests of the patient and to provide against the dangers of infection to his household and associates. Careful and detailed instructions are given as to ventilation and the principles of the fresh-air treatment, and practical demonstration is made of the proper method of disinfection of the bed clothing, eating utensils and personal linen of the patient; also any necessary suggestions are made along the lines of general cleanliness and better hygiene. Thorough inquiry is made concerning the care which the patient habitually takes of his expectoration, and notice taken of the general attitude of the whole household toward the instructions given at the clinic. Investigation is made into the available facilities for taking a " rest cure " out of doors, and the fire-escape, the roof and the nearest park are all considered from this point of view, and the results incorporated in the report. For a few patients we have been able to secure sleeping bags, reclining chairs, or hammocks, so that they may stay out of doors almost continually. The family resources are also noted and recommendations for assistance are made accordingly. Oftentimes the " home " is absolutely unsuited to our purpose; if so, it is so stated, and the actual conditions existing are described. It is easy to see how indispensible the visiting nurse is to the physician who would manage his case intelligently.

3. *The education of the patients* and their families in the principles of good hygiene and the prevention of the spread of the infection. How this is accomplished by the constant reiteration of sound advice from physicians, nurses and printed circulars, is apparent. During the past four months our nurses have visited 138 patients whose aggregate families number 621. Each one of these individuals is now a centre of information in regard to the general principles of healthful living.

4. *The study of social and economic conditions* obtaining in each case. This means the correlation of all the available data obtained from the investigations of the physicians and of the nurse. Upon this study the physician bases his method of management of each case. This involves upon the part of the physician an intimate knowledge of all available resources. In our work we keep in close communication with all the sanatoria and hospitals which admit tuberculous patients and attempt to send suitable cases to the institutions best adapted to their needs. The regulations of the Health Department and Tenement House Department are studied and observed; the facilities offered by the Charity Organization Society, the United Hebrew Charities, the Association for Improving the Condition of the Poor, the various churches and religious societies, the Diet Kitchen Association and the Strauss milk depots are all well known and constantly

utilized. In addition we distribute milk and eggs from the hospital store free to all patients who are found to be unable to sufficiently provide for themselves. Lately we have erected a tent cottage upon the hospital grounds, accommodating twelve patients, which is for the use of such patients in the clinic whom it is desirable to keep under observation temporarily, or for favorable cases for whom proper sanatorium or home treatment is not possible. The proper use of all these means is an essential portion of the treatment, and the responsibility for it rests with the physician, from whom it requires careful study and judgment. It is the lack of just this which renders the ordinary dispensary treatment of these cases so inadequate and unsatisfactory.

5. *The opportunity for scientific investigation* and clinical experience for physicians and students. This portion of the work has not yet been developed. It is proposed to take it up systematically next autumn, and it is hoped that tuberculosis will be studied in this clinic along various lines.

REPORT OF THE TUBERCULOSIS DIVISION OF THE BELLEVUE HOSPITAL OUT-PATIENT DEPARTMENT.

From January 1 to May 31, 1904.

Number of new patients treated	210
Number of visits to clinic by all patients	850
Number of visits by nurses in the homes	593
Number of cases untraced	39
Number of patients sent to hospitals	89
Number of patients under observation May 1	91
Number of patients given material assistance	139

Summary of Material Assistance.

SOURCE.	NUMBER OF PATIENTS.	ASSISTANCE.	
		Quarts Milk.	Dozen Eggs.
Bellevue Hospital	35	920	685
N. Y. Diet Kitchens	19	1,836	....
Strauss Depots	8	570	....
Charity Organization Society	37	General Relief	
Ass'n for Improving Condition of the Poor	16	"	
United Hebrew Charities	4	"	
Miscellaneous Organizations	20	"	

Total milk given, 3,326 quarts—cost	$166 00
Total eggs given, 685 dozen—cost	144 00
Total cost or milk and eggs	$310 00

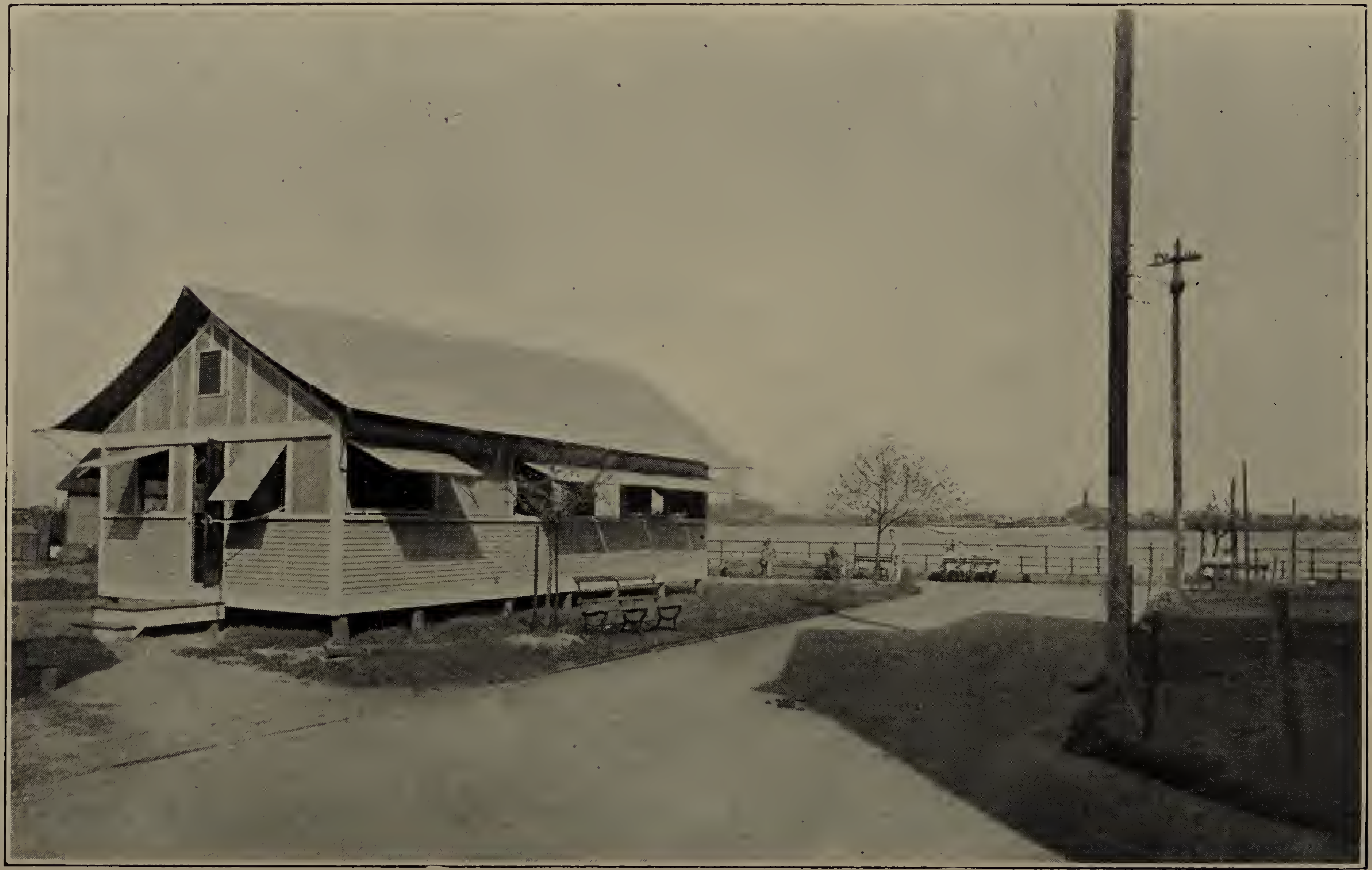
It will be noted from the above report that of the 210 patients treated, 39, or 19 per cent., were untraced in their homes. These are almost all lodging-house cases, whom it is very difficult to manage from the dispensary. They should all be sent to hospitals, if not willingly, then compulsorily, by authority of the Health Department.

It will also be seen that 89 of our cases, or 42 per cent. were sent to hospitals. These were advanced cases and others, such as lodging-house cases, who were unable to have proper home surroundings. We consider this a very successful portion of our work as it has removed foci of infection from the city, which, under the circumstances, are inevitably dangerous.

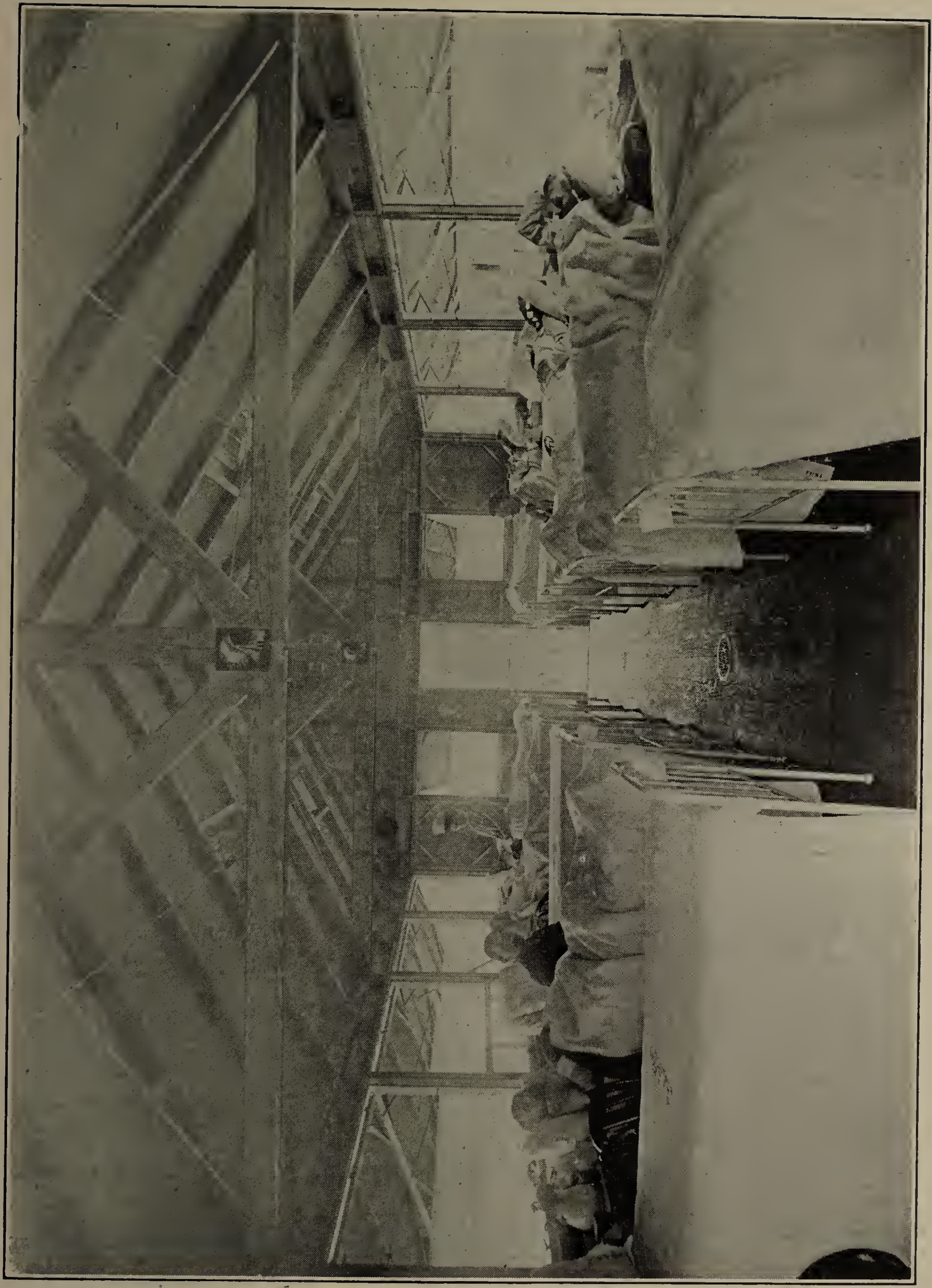
Attention is also requested to the cost of the food dispensed under our direction, as a portion of the treatment. This was $310. This means that for this sum we were able to assist every patient who was found after careful investigation to be unable to adequately provide the necessary extra diet for themselves. We consider this a very moderate amount, and it demonstrates the advantages of thorough investigation of these cases by trained workers. Indiscriminate giving of food, even for treatment, would be very pernicious in the moral effects upon these patients and their families.

Our deep indebtedness to the different charitable organizations is also very apparent from our report. This is especially true of the Charity Organization Society, the New York Diet Kitchen Association and the Strauss Milk Charity. Much of our work would not have been possible without the co-operation of these organizations, and it is with deep pleasure that we acknowledge our obligation to them.

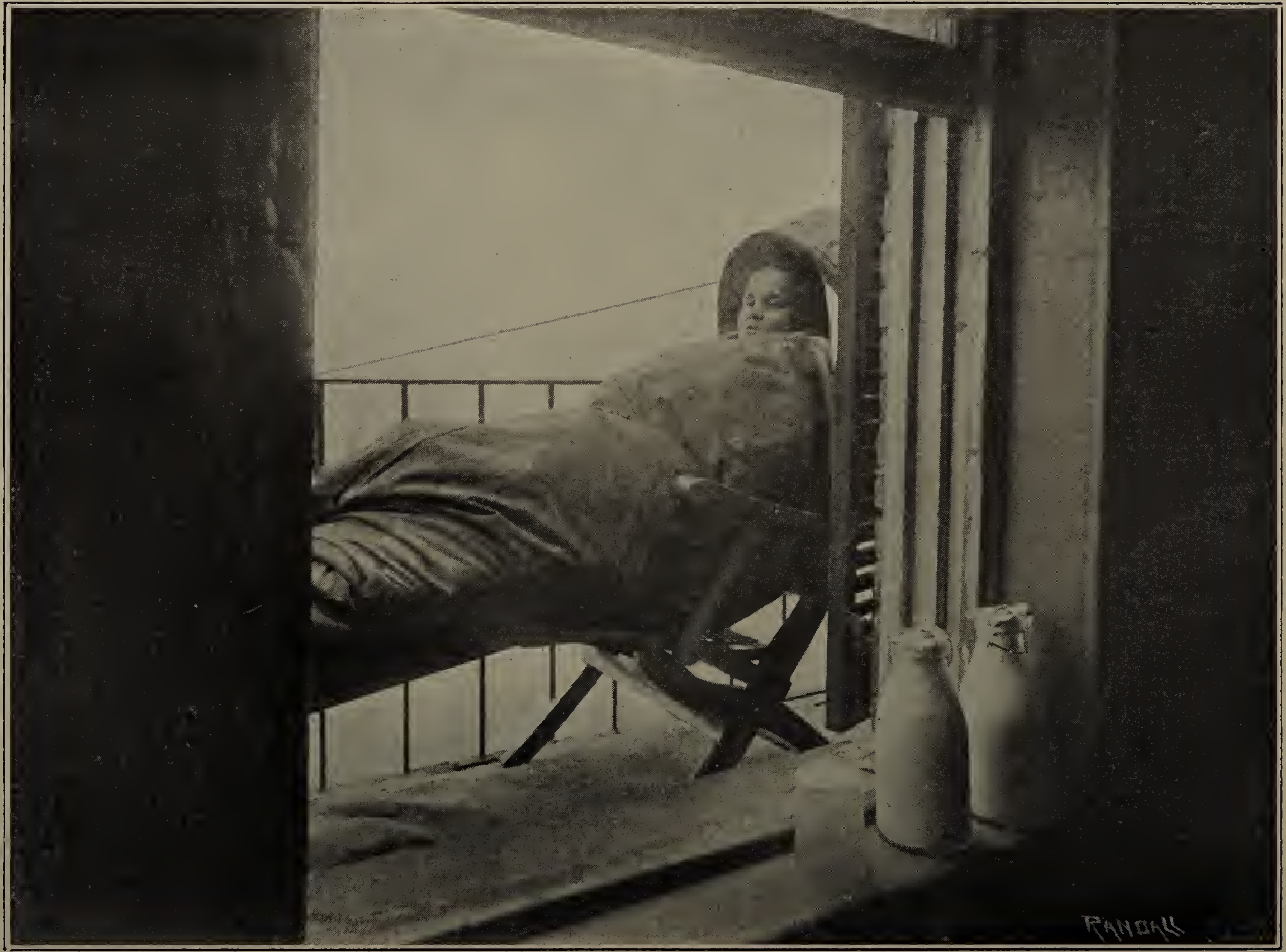
The successful establishment of this clinic is due very largely to the very able assistance of Doctor Haven Emerson, the late Doctor E. L. Trudeau, Jr., and the visiting nurses, Miss Hopkins and Miss Damer. It is more especially attributable, however, to the deep interest and vigorous support given to it by Doctor John W. Brannan, President of the Board of Trustees, and by Doctor Mabon, Mr. Rickard, and Miss Delano, Superintendents of the Hospital and Training School.



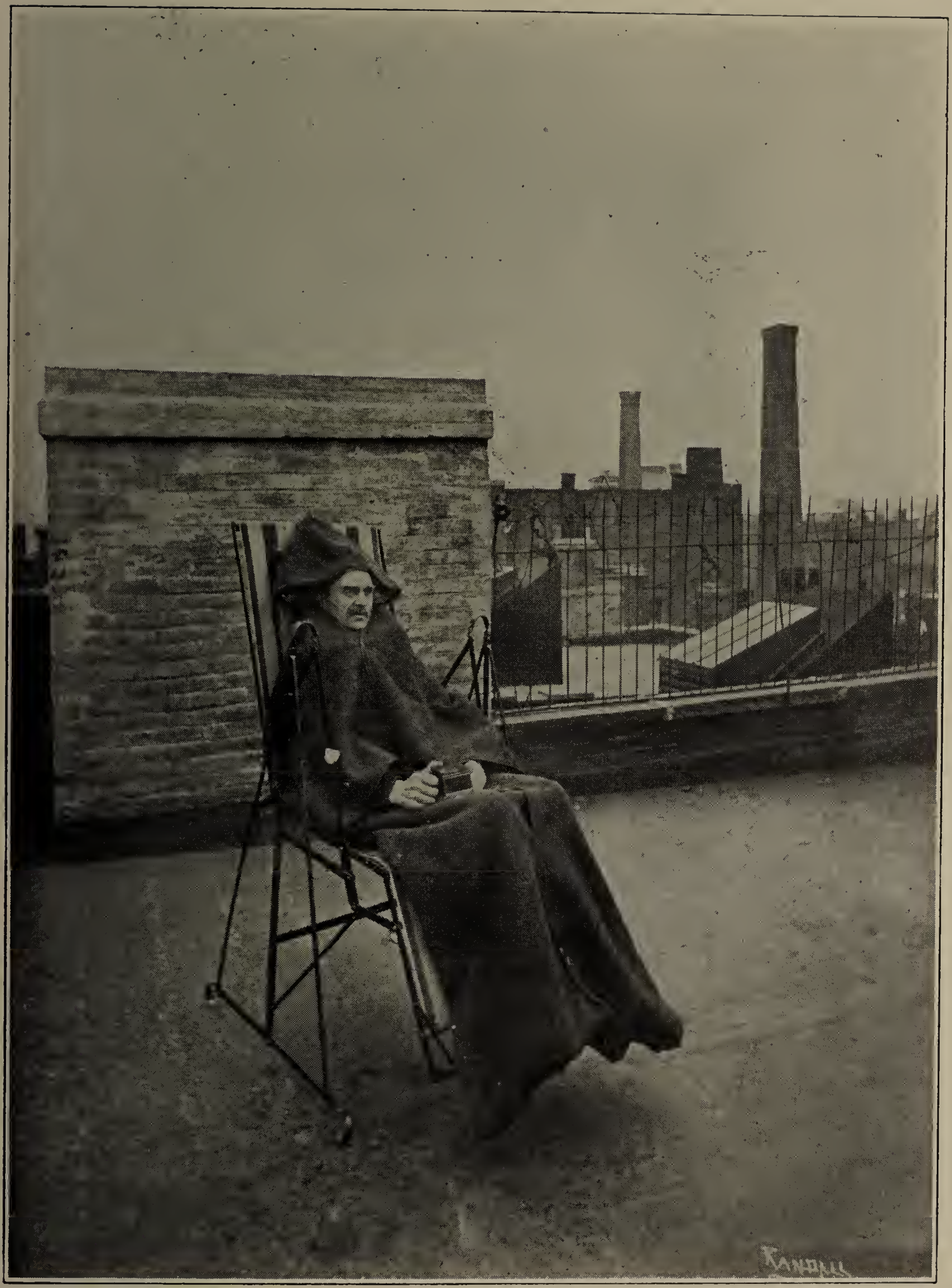
1. The Tent Cottage. Showing the Outlook upon East River.



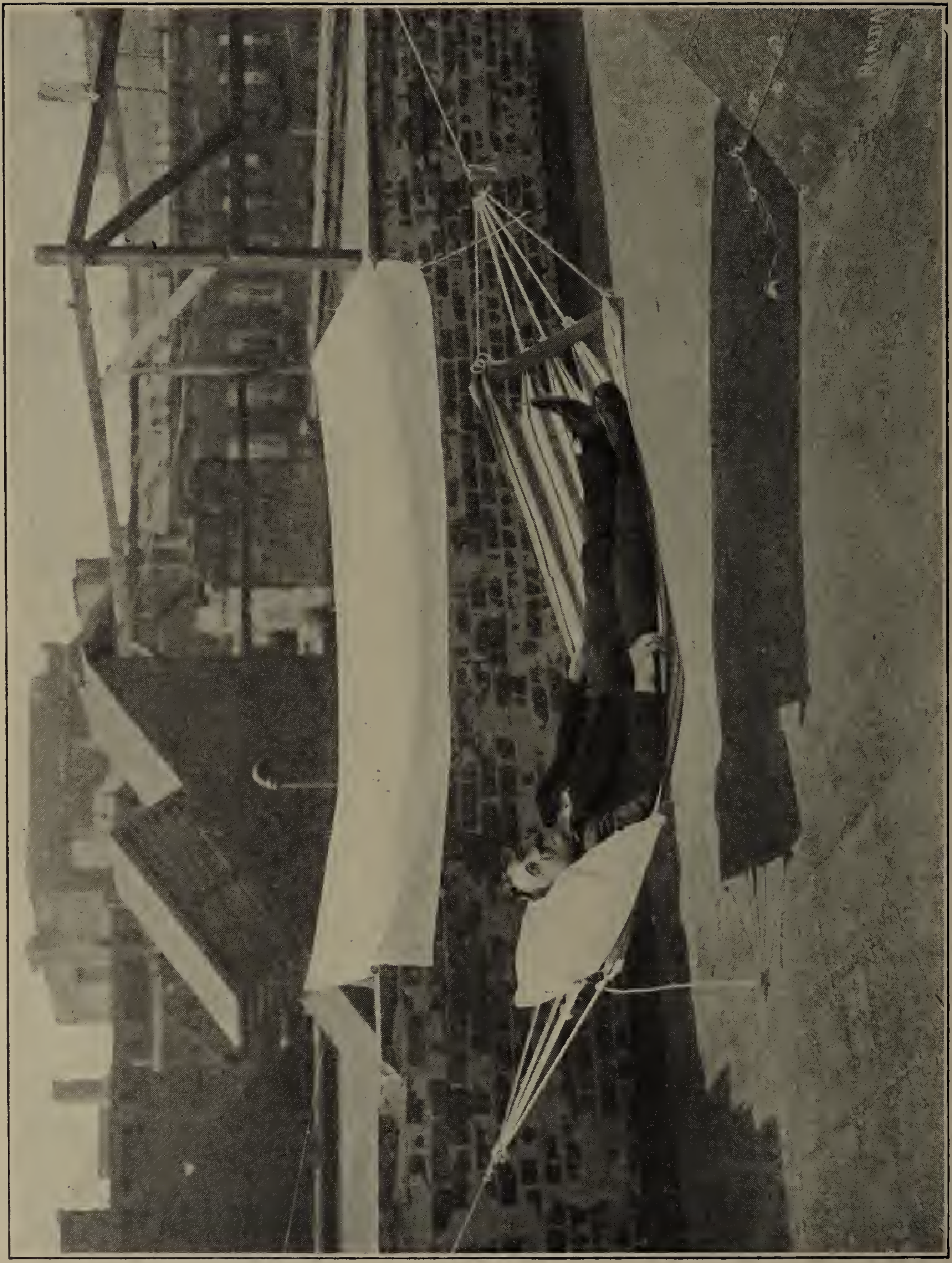
2. Interior of the Tent. It is lighted by gas and heated by steam.



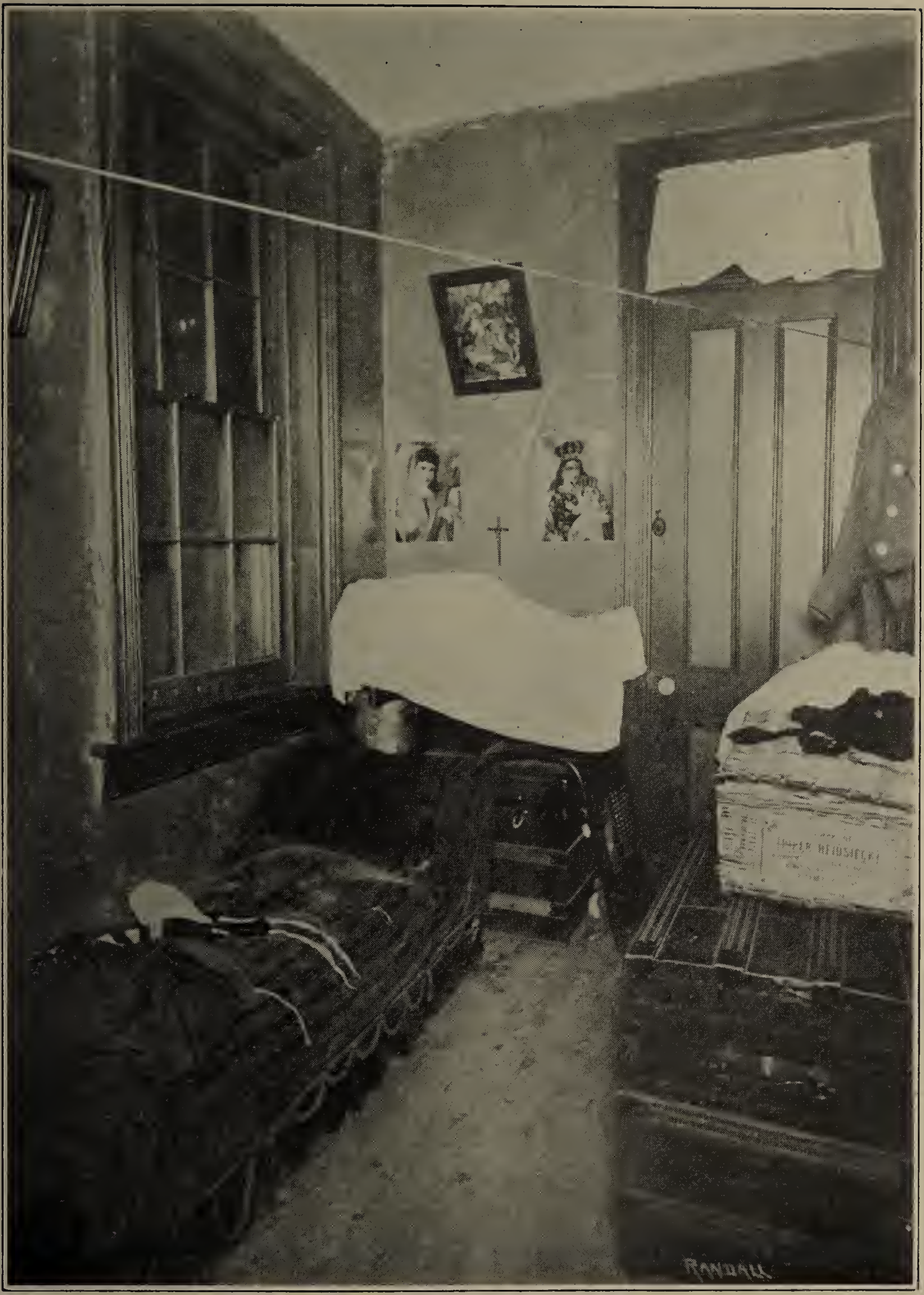
3. Fresh Air Treatment upon a Tenement Fire-escape. This Patient Gained 16 lbs. in Six Weeks.



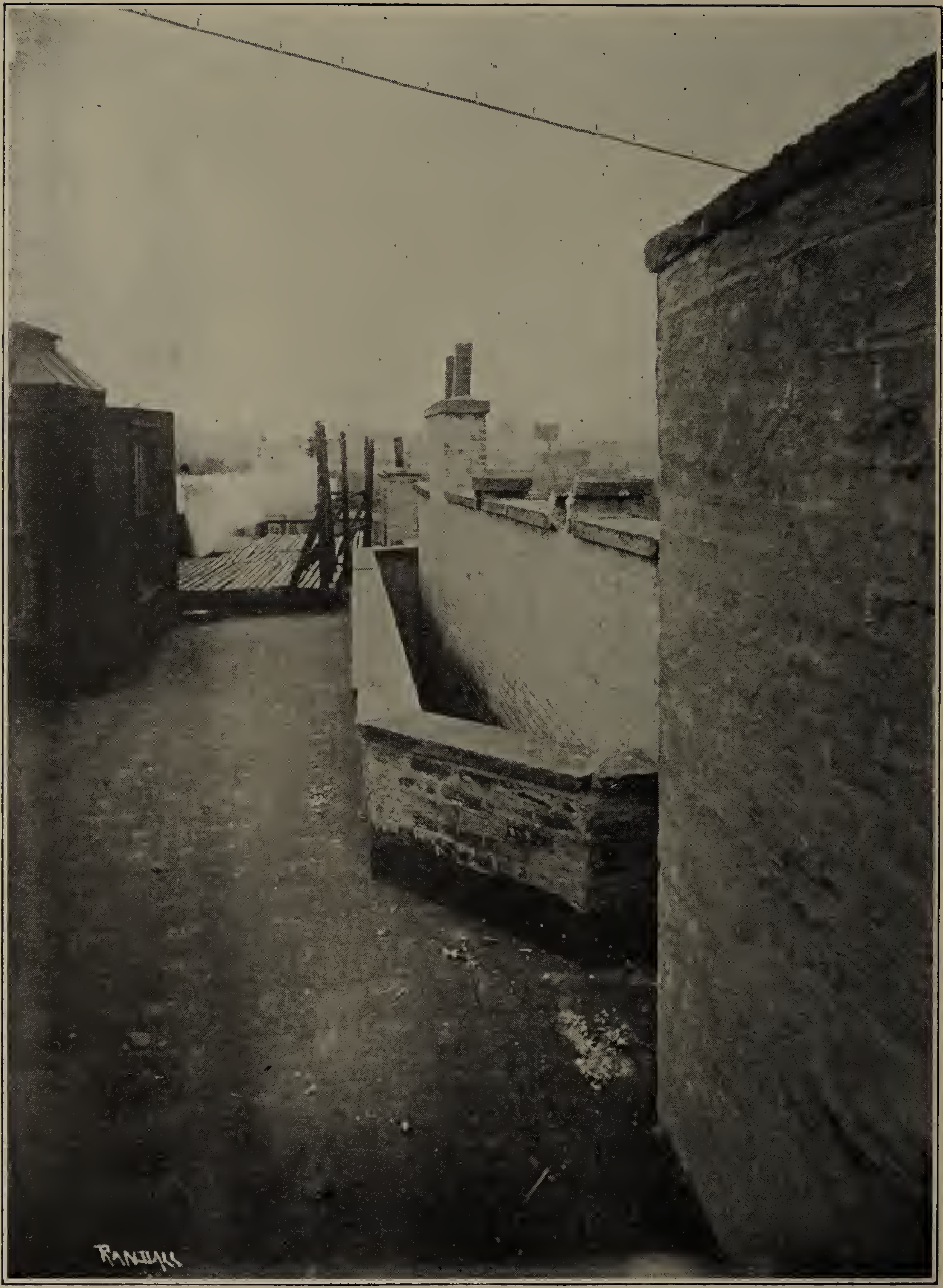
4. Fresh Air Treatment upon a Tenement Roof.



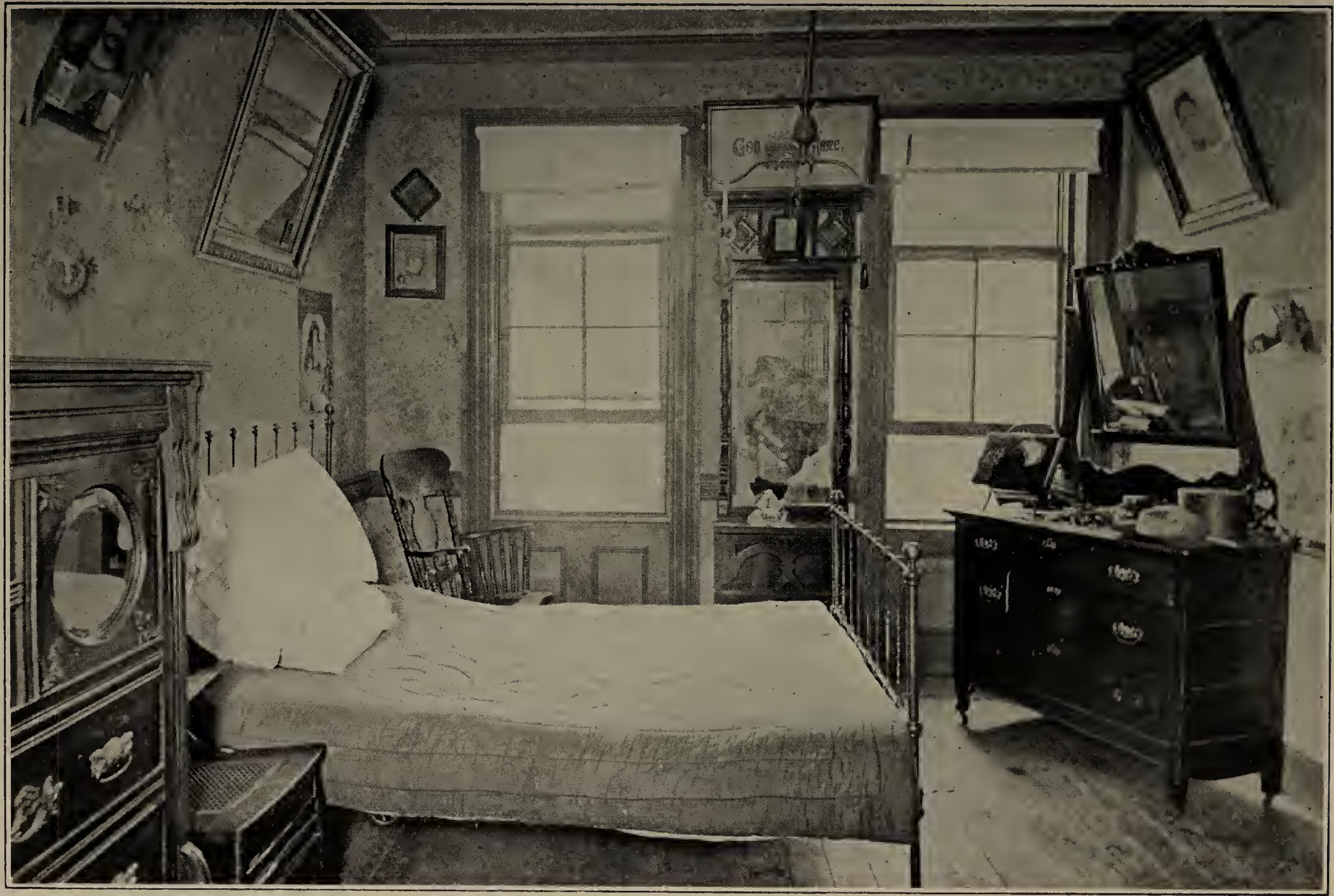
5. Another Device upon a Roof.



6. An Inside Dark Room in which one Patient was found to be Sleeping.



7. The "Air Shaft" supplying the same inside room.



8. Large, Airy Room into which this Patient was moved by the Nurses' Direction.

REPORT OF THE TUBERCULOSIS CLINIC AT GOUVERNEUR HOSPITAL.

From October, 1903, to July, 1904.

STELLA S. BRADFORD, M. D.
N. GILBERT SEYMOUR, M. D.

Read before the Section on Medicine, New York Academy of Medicine, May 17, 1904.

Before October, 1903, there was no separate clinic for tuberculous patients in any of the hospitals under City jurisdiction. In Gouverneur, as in many other hospitals, these patients were treated in the general medical clinic. The diagnosis of the cases reported to the Board of Health was based chiefly on the history and general appearance of the patient, confirmed by a more or less thorough physical examination. A sputum examination was rarely made. Little attempt could be made to instruct the patients in the care of the sputum nor in hygiene. The treatment consisted chiefly of Stokes' expectorant, cod liver oil and stimulants. It is hardly necessary to say that this condition of things was due to the exigencies of the situation rather than to any negligence.

Last October we were asked to differentiate the tuberculous cases at Gouverneur Dispensary, and, if possible, to build up a clinic. We were told that there was, at that time, hardly more than one case a day. In January we received our formal appointments.

Considering the fact that a separate clinic has been in existence but nine months, we shall not attempt an account of scientific investigation of obscure symptoms, nor a discussion of the relative values of different methods of hygiene and medication. We shall report our observations in a series of 115 cases.

The exact conditions obtaining at present in the Gouverneur tuberculosis clinic are as follows: It is held three times a week—Monday, Wednesday and Friday, from 11 to 1—in the dispensary in the basement of Gouverneur Hospital. In the waiting-room our patients assemble with those of the general medical and nose and throat departments. There is but one room available for history taking, physical examination, prescribing, weighing, and instruction in hygiene and breathing exercises. During the first part of our period a general medical clinic for women and children is in progress; and during the second, an active nose and throat clinic, the two usually overlapping, so that not infrequently there are fifteen to twenty people in the same room at once. But during the last part of the period we are able to use the adjoining rooms for examination. All the rooms are below the street, which is cobbled, and a thoroughfare for heavy trucks. We are, however, fortunate not to be on a trolley line, and, during the winter, the snow is as grateful to us as tan bark to a fever patient.

During the eight and one-half months, from October to June 20, 115 patients have been entered and ultimately proved to have tuberculosis. Of this 115, 75 were males and 40 females. The range of ages is very large, including all decades from the first to eighth; the largest number, however, belonging to the fourth and fifth. That is, we have enrolled 33 between thirty and forty years of age, and 33 between forty and fifty. The nationalities represented are chiefly Russian, American, Irish, Austrian and German, but there are also representatives from Australia, Canada, England, Hungary and Norway. The United States is second only to Russia, the latter country being the birthplace of 53, while 40 were born in this country.

In regard to the character of the cases, it is our first impulse to say that we have admitted but few which are advanced. To be exact, 18 might properly be called incipient, 60 active, 27 advanced, and 10 quiescent. The larger number of those giving intelligent histories described their disease as existing for a term of years averaging 5½, while those who claimed to have had no symptoms till within a year show an average duration of four months. In a series of 22 patients who could tell with fair accuracy their previous weights, the average loss before coming to the dispensary was 24½ pounds, the greatest loss being 55 pounds and the least 7.

It may be interesting to note the symptoms given by the patient in applying for treatment. Among the chief complaints on admittance, cough was given 89 times; weakness, 41; pain in the chest, 25; hæmorrhage, 13; and following these in frequency, digestive disturbances, dyspnœa, headache, hoarseness, nervousness, night sweats, and afternoon temperature. In general, the symptoms were fairly classical. Cough was without exception the most common, existing to a marked degree in all but four cases. Next in order are digestive disturbances, existing in 74 cases; 59 gave a history of hæmorrhage, which, in 30 instances, was hardly more than blood-streaked sputum; 59, a history of dyspnœa; and 45, a history of night sweats. Almost without exception, there was a marked loss of flesh and strength.

Our diagnosis for the report to the Board of Health is based upon the composite of history, physical examination and sputum examination. For six months we made our own sputum examinations; but recently, owing to increase in numbers, we have referred them to the Board of Health. Of the sputum examinations, 54 per cent. have, so far, been positive.

Our system of treatment is a very simple one, and is the result of a process of evolution, in which the grade of intelligence of the patients and the facilities at our command, were important factors. Most of our cases are referred from other departments, though some are brought by friends and some are sent directly by the dispensary clerk. A full history is taken on the printed blanks recommended by Dr. Miller and provided by the City hospitals. A thorough physical examination, including chest measurements, follows. The patient usually asks what his disease is, and is told that he has some trouble with his lungs, that examination of the sputum is necessary, and that, in any case, if he follows directions, he can be much better than he is at present, and may possibly become entirely well. Usually he complains of constipation and no appetite. Therefore he is given an initial dose of calomel and salts. Almost invariably he complains of cough. Therefore he is given heroin, which we have found so satisfactory in the treatment of this symptom that we have used it almost exclusively. The patient is then told to spit only in the sputum cups which we provide—which have been supplied to us by the Charity Organization Society and the Board of Health—and to return to us in two days with a specimen of his sputum. His condition at this second appearance is almost always improved. His digestive tract is clear, his appetite better, his cough relieved, and he has slept. In consequence, his spirits have risen and he talks of going to work again. It is then that we emphasize the fact that, though he has made a good beginning, the process will be a long one. Then begins the progressive instruction in hygiene, which is continued and repeated during many weeks. As rapidly as the patient's mental capacity will allow, he is told of the necessity for pure air, of the kinds of food to use and those to avoid, and of the bad effects on his present condition of alcohol and tobacco. He is taught breathing exercises, in which we have been glad to follow the methods advocated by Dr. Knopf, and used by him in the Riverside Sanatorium. Prophylaxis for others is gradually taught. The average patient is told but few of these things on his second appearance, and chiefly those relating to air and diet.

Where stimulants or tonics are needed, we depend almost wholly on strychnine, given in the form of the tincture of nux vomica, m. v. to xv., three times a day, usually administered in a vehicle of the mixture of rhubarb and soda. When a further laxative is needed, especially in the constipation attendant upon a milk diet, we add to this combination cascara in a dosage found to be appropriate for the individual. Special symptoms we have treated as they arose. The most common, bloody sputum, has usually yielded to calcium chloride, given in aqueous solution, in a dosage of 10 grains every four hours. The few who have reported extensive or repeated hæmorrhages have been ordered to remain at home and in bed for a time, or have been referred to hospitals.

Two other therapeutic agents which we have used regularly are cod liver oil and ichthyol. In comparing the cases treated by these two we find the results surprisingly similar. Ichthyol was administered in aqueous solution, beginning with a dose of 5 minims and gradually increasing to 25 minims, three times a day. It has been almost without exception well tolerated, and there have been no ill effects. Cod liver oil was given in emulsion. Some patients who did poorly on one method were transferred to the other. It seems evident that they were unsuitable for either, since they did not improve under either method.

One of the most interesting observations we have made has been concerning the value of heroin in controlling cough, a symptom so distressing that unless it is relieved the patient becomes discouraged and is likely to go elsewhere. We have throughout avoided the administration of cough mixtures containing a number of ingredients or a sweetened vehicle. We have, therefore, relied almost exclusively upon heroin, given in aqueous solution, beginning with a twenty-fourth of a grain four times a day, and increasing or diminishing the dose and frequency according to individual indication. With few exceptions it has proved satisfactory, and there have been no ill effects from its long continued use. In obstinate cases, where several remedies have been resorted to, as codeine and Stokes' expectorant, it has proved ultimately the least objectionable and the most efficient.

All cases with laryngeal involvement have been treated in the nose and throat clinic already mentioned.

In general, our records give these further statistics. Of the 115 cases, 69 are at present (June 20) being treated with fair regularity in the dispensary. Ten are in hospitals, 2 have gone to the country and 1 to a "private doctor." Of the remaining 33, many have been referred to hospitals, but we have as yet no means of learning whether they have gone as directed, or whether they were accepted. Of the whole number, 31 have gained weight, the average being 4 pounds, and the greatest gain 17 pounds; 16 have lost, the average being 2¼ and the greatest loss 7 pounds. Owing to the crowded condition of the room, it is impossible to weigh the patients stripped, and in consequence, the weights vary with the amount of clothing.

Our difficulties in treatment have been twofold. In the first place, until recently we have had no way of providing our patients with milk and eggs. Since the hospital has been providing these "on prescription"—that is, for two months—there has been an appreciable gain. Secondly, we have had no visiting nurse, and hence have not been able to care for the patients during exacerbations, nor has it been possible to follow up those cases which have failed to return with regularity. We have used postal cards as a poor substitute, and in this way have recalled many who had failed to report.

Of course, it is not as though we were dealing with simple tuberculous infection in otherwise healthy human organisms. Fifty-five of our cases had complications of more or less gravity. Among these were 14 with organic or functional heart disease; 7 with arterio-sclerosis; and there were also cases with adenoids, enlarged tonsils, rheumatism, gout, chronic nephritis, diabetes, hernia, pregnancy, oöphoritis, menorrhagia, prolapsus uteri, tabes dorsalis, paralysis agitans, early syphilis, epithelioma of the lip, and carcinoma of the stomach.

Moreover, the habits of our people are not such as to increase their resistance. Bathing and exercise are almost equally unknown to them. Fifty-nine use to excess tea, coffee, alcohol or tobacco, or all combined.

Their occupations are about as bad as possible from the point of view either of the patient himself or that of his associates. Only 15 are in suitable occupations—that is, are employed as messengers, drivers, caretakers and peddlers; 42 are in occupations which may be suitable if there is intelligence in hygiene and prophylaxis. This second class comprises school children, housewives and day laborers. The third class, numbering 46, are in occupations endangering the patients themselves, or the community, or both—that is, they are barbers, tailors, butchers, cooks, waiters, factory hands and longshoremen. Nevertheless, fully 50 per cent. of our cases claim to be able to work and are in need of suitable employment.

The intelligence of these patients varies greatly. Some at once sense the situation and work with us, reminding us when their sputum cups need replenishing, and bringing in their friends who seem to them to need treatment. Among our most intelligent are the school children, who take kindly to the idea that they are in training as important as that of any athlete. On the other hand, there are those who come when they feel the need and stay away at the slightest improvement. Also, we must take into consideration the status of the man who with equanimity upset his sputum bottle in his pocket, and the woman who, on being told to hold her hand before her face when she coughed, promptly spit into it!

We have no exact data in regard to the social conditions represented. Of one thing we are sure—that the medical profession is not being impoverished because these people are treated free. Occasionally we have a well-dressed child, but the appearance of some other member of the family outweighs the first impression of competence.

Neither have we accurate statistics in regard to the sources of infection. We know that 31 gave a history of consumption in the family; that 19, in like manner, gave a history of cough or asthma; but of the 65 with negative history we know nothing. Yet we are often told that "everyone coughs or spits on the floor" in the shop or lodging-house, as the case may be. It is evident that investigation of such sources of infection is of the greatest importance.

Our contributions to the education of the public have been indirect. We have distributed very little literature. It has seemed to us that what the patients know they will talk about, and that those with whom we work are usually more confused than helped by printed information. It is obvious that this is not universally true.

That the clinic is growing is attested by the fact that, whereas in October we treated 1 to 2 cases a day, we now treat 10 to 20, and in one month we have enrolled as many as 24 new cases. Also, we are assured by Dr. Brannan that there will be ample provision for the tuberculosis clinic in the new Gouverneur dispensary in the west wing of the hospital, the work upon which has already begun.

After a little experience and a little reading, it is easy to construct in imagination a dispensary which seems ideal. Practically, space and quiet are the two most obvious essentials. The building should, of course, preferably be clean, airy, warm and abundantly supplied with spittoons. The waiting and history-taking rooms should be adequate to the size of the clinic. The examining room should be light and quiet. In addition, rooms for hydrotherapy, breathing exercises and sun baths would contribute materially to the equipment. To make the work really efficient nurses are essential, to report the financial condition of the patients, unreported cases in the neighborhood, the intelligence of patients in carrying out instructions, to care for patients during exacerbations, and to assist in the dispensary on clinic days.

There are two important channels open to private charity or to civic wisdom. The first will provide methods for caring for the children of consumptives, thus enabling the parents to go as soon as necessary to the hospitals; and, what is even more important, so training the children in hygiene that their tuberculous predisposition may be overcome. The second is concerned with providing work for many who are willing and able to do suitable work. We have among our patients enough to insure good workmen were some of the lighter positions in the care of the parks and public buildings reserved for their competition. Many would make good peddlers, watchmen and messengers. Our work will not be adequate if, in preserving a man's existence, we do not also preserve his activity—that is, his life.

June 20, 1904.

CONCERNING THE ANATOMY AND TRAUMATIC ETIOLOGY OF ABDOMINAL HERNIA.

By B. B. Gallaudet, M. D.

This paper contributes nothing "new" to what is really known about these aspects of hernia.

It is simply a series of comments, suggestions and explanations on and about what others have written; and is offered with the hope of clarifying some of the statements in these writings which are at least conflicting, and which, occurring as many of them do in various text-books, cause confusion in the minds of students for whom these books are primarily intended.

In the first place, as to the **anatomy** of abdominal hernia:

What others say: Most writers define abdominal hernia as a "protrusion *through*" the abdominal wall (the "opening" in the muscle layer being a normal one or acquired) of a viscus or a portion thereof, said protrusion being "always" covered with skin and fascia and by the "sac," *i. e.,* an investing pouch of peritoneum. In other words, an abdominal hernia consists of: (1) "contents" (the viscus or organ or portion thereof); (2) the "sac"; (3) the "coverings" (*i. e.,* all structures other than the "sac") which cover the "contents."

Comment as to the wording of this definition: If the "protrusion" is "through" the "abdominal wall," it should be "through" the peritoneum also, and then there would be no "sac"; and it should be "through" the skin and fascia also, and then there would be no "coverings"; and the final result would be a "penetrating wound of the abdomen," with "protrusion of viscera," and not a "hernia" at all. Hence the necessary proviso in "what others say," that said protrusion is always covered with skin and fascia and by the "sac." It is understood that the "opening" of the sac is *not* an opening *in* the peritoneum; "opening *into*" the sac, would be a better expression. Since a proviso always weakens a general definition, it is advisable to do away with it if possible; and preferably by changing the cause for its existence; which in the present instance is the use of the term "protrusion *through* the abdominal wall."

Suggestion: An "abdominal hernia is a protrusion, *in* the abdominal wall, of a viscus or a portion thereof, through an opening (normal or acquired) in the muscle layer only." Then should follow an explanation of the "sac" and "coverings," whose existence is *implied* in the statement that the "opening" is in the *muscle layer only.*

Comment as to the correctness of the statement that an abdominal hernia "always" has a "sac":

Without wasting words, this means, can an abdominal organ, or part of it, ever occur lying *in the tissues* of the abdominal wall without any peritoneal covering, *i. e.*, without a "sac" (but, of course, covered by skin and more or less of the fascia) and also be a *hernia?*

That this anatomical condition does occur is acknowledged probably by every one nowadays. The trouble is, however, that many writers do not consider it a "hernia," call it a "traumatic rupture," and claim that it is *always* caused by "traumatism," meaning a blow or crushing force only.

This statement is unfortunate in three respects: First, it is, broadly speaking, not true; second, in not calling this condition a "hernia"; and third, in regarding "the traumatism" as consisting of only a "blow" or "crushing force." As a matter of fact, this is simply a *form* of traumatism, *i. e.*, "external violence," the other form, which is ignored, being "internal violence" or "muscular action." Now, to answer the original question: A hernia without a sac can exist; that is, one caused by "muscular action" only, like other herniæ; and the anatomy of which is exactly the same as that of the "traumatic rupture" just referred to. Therefore, if a condition of viscera *in* the abdominal wall, without a "sac," and caused by traumatism (muscular action) is a "hernia," there is no reason for calling an exactly similar condition, also caused by traumatism (external violence), a "traumatic rupture"; or for calling it anything else than a "hernia." In fact, just as in etiological classification of other abnormal conditions, both of these conditions should be called "traumatic hernia," and then the difference stated as to *cause.*

Another reason for not using the term "rupture" in this connection is that it already possesses the distinct meaning of a tear or " wound " of an abdominal organ occurring without the existence of a " wound " of the abdominal wall; in other words, " rupture " of abdominal viscera is a form of " abdominal contusion "; under which term, it may be noted, should it ever seem desirable not to use " hernia " at all in their connection, could be grouped both of these conditions. But, whatever they are called, they should be treated alike, since the only difference is, as already stated, in the *causes:* of one, " external violence," and of the other, " internal violence " or muscular action.

The statement already made, that a hernia " without a sac," and not caused by " external violence," can exist, is to be found in very few, if any, text-books; but confirmation lies in the author's own experience, which embraces one of these cases.

The case: A man of marked muscular development.

The history: Signs and symptoms were identical with those of the ordinary " strangulated inguinal hernia," *i. e.*, the " swelling " of moderate size came on suddenly, after a violent muscular effort. No " external violence," and " no hernia " previous to the effort. Operation revealed a coil of small intestine, moderately congested, lying in the inguinal canal and with " *no sac.*" Around the margin of the " internal ring " could be made out the torn edge of parietal peritoneum, which, after reduction of the intestines, could be readily stretched and sewed together. Uneventful recovery.

Further comment: A " hernia without a sac " and not caused by " external violence," really comprises *two* subdivisions; the first has just been described. The second is mentioned in a good many text-books, and is called " hernia without a sac, or with only *a partial sac.*" Examples are given in which the *cause* is muscular action acting slowly; the " contents " cæcum or bladder, and the region the *inguinal canal.* The latter part of the definition, *i. e.*, " with only a partial sac," does not seem of possible occurrence. Nor does it seem clear, from reading the descriptions of these herniæ, that the authors really had this idea because of the prominence given in these descriptions to the fact that the organ concerned, owing to its partial investment of peritoneum, slides under the adjacent parietal peritoneum, and thus, with or without stripping off from itself some of the peritoneal covering it possesses, presenting itself at the " internal ring," passes through this into the inguinal canal.

This explanation is undoubtedly correct; but this procedure can not produce a "partial sac," because the viscus slides under, and thus lifts up from the "internal ring" the parietal peritoneum normally stretching across that "aperture." If it did anything else it could only do one of two things—1: Go through this peritoneum; or, 2: push it ahead; and in either event it would not be this kind of a hernia.

To return: What does happen is this: As the partially invested (by its own "visceral" peritoneum) viscus, or portion of it, descends into the canal, it drags after itself a "tube" of parietal peritoneum, either simultaneously, or after it ceases stripping off from itself its own visceral peritoneum; the "bottom" of the "tube" being visceral peritoneum *on* the organ; unless the "bottom" were formed by the walls of the tube being "pinched together" by adhesions just above this visceral peritoneum. It may be that this after-coming "tube" is what is meant by a "partial sac." If so, the term must be a misnomer, because a "sac," even if "partial," must inclose something; while in this case the organ or main part of it lies in the tissues of the canal entirely uninvested. Whatever is left on it of its own peritoneum, although in direct continuity, by *reflection*, with this after-coming "tube," naturally does not count as a "sac," or portion of one, or as anything else which might, even partially, "inclose," any more than does the visceral peritoneum of, and completely investing, intestines in any ordinary hernia.

It is assumed that such a hernia takes place in a normal canal, *i. e.*, in one which is *not* the site of a previously formed "sac"; which combined formation would of course be possible; but then the relation of this viscus to this "sac" would be the same as that of the viscus to the "sac" in an ordinary infantile hernia, viz.: "retro-funicular."

Suggestions: As to the name of these two kinds of "hernia without a sac," why not call them "interstitial" herniæ? The term would be appropriate, because it would indicate the fact that the organ does lie among the "interstices" of the surrounding tissue; and another reason would be that this use of the term would prevent its use in many text-books, meaning a hernia with a "sac," but with the sac placed properitoneally and between various combinations of the layers of the abdominal wall; as if it made any difference where the "sac" is, from a major terminological point of view, as long as there is a "sac."

Furthermore, the term "properitoneal," properly modified to indicate the position of the sac, for such herniæ would be exact enough.

To distinguish between these two forms of "interstitial" herniæ: The first, having no "attachment" of parietal peritoneum, could be called "*free* interstitial hernia." The second form, to repeat, which drags after it a process or "tube" of parietal peritoneum, with which its own partial covering of visceral peritoneum is in continuity by surface reflection, and to which it is thus "attached," could be called "*attached* interstitial hernia."

Returning now to the *original definition,* it will be seen that it applies only to herniæ with a "sac"; and there is still lacking a definition which will apply both to herniæ with a "sac" and to "interstitial herniæ" (herniæ without a sac). The following is suggested: An abdominal hernia is a protrusion *in* the abdominal wall, of a viscus, or portion thereof, through an opening (normal or acquired) in the muscle layer *only;* or, in the muscle layer (normal or acquired) *and* the peritoneum (acquired). As already stated, "opening" *into* the "sac" must not be confounded with "opening" through or *in* the peritoneum.

This definition, of course, does not include certain *bulgings* of the abdominal wall (often called herniæ) in which there is no "opening," even in the muscle layer, which is simply stretched over the viscus and its sac like the skin and fascia. It is questionable whether these conditions should be called herniæ at all. It is suggested that they be grouped under abnormal conditions of the abdominal wall and called "prolapse of the abdominal wall."

In November last (1904), during a term of his service as Visiting Surgeon to Bellevue Hospital, the writer performed a herniotomy for an apparently regular reducible inguinal hernia; which proved to be what has just been described as "attached interstitial hernia."

The Case: Male; middle-aged; healthy; and with marked muscular development.

The "hernia" was *left* inguinal, extending about half way down the scrotum; of slow growth—two or three years; with no history of "external violence" as either direct or predisposing cause, or of muscular action as direct cause; and presented the usual signs and symptoms.

The operation showed the following conditions: (1) The "contents": The viscus was a loop of the sigmoid flexure; (2) Relation of peritoneum: Of this protruded intestine the *inferior* and *external aspect* was entirely uncovered by peritoneum and was equal to somewhat less than one-half of the circumference; the *superior* and *internal aspect* completed the circumference and was covered with its normal "visceral" peritoneum, under which was seen (as is usual) one of the tæniæ; (3) The "sac": This was a "tube" of parietal peritoneum with relations to the peritoneal cavity and general parietal peritoneum exactly like those of the "sac" of an ordinary hernia; the "bottom" of this "tube," however, was the superior and internal aspect just described of the loop of intestine; that is, right over this aspect was reflected, as visceral peritoneum, the lower end of the "tube" of parietal peritoneum.

The operation was continued by simply suturing the incision made in the "sac," and returning the "tube" and protruded intestine into the abdomen; very easily accomplished after incising the edge of the "internal ring," through which it was perfectly evident the hernia had come.

The final steps were those of a Bassini herniotomy, modified, as is the writer's custom, by suturing the aponeurosis of the External Oblique *behind,* or "under," rather than *in front of,* or "over," the spermatic cord. Uneventful recovery.

As to the **traumatic causation** of abdominal hernia:

The expression "traumatic hernia," although meaning simply a *hernia caused by traumatism,* is often misleading: (1) because there is rarely any distinct statement as to which element of the hernia, "contents" or "sac," is the one so caused; an important consideration, for the reason that each of these elements may have its own cause, different from that of the other; (2) because there is often no distinction made as to whether the traumatism is "external violence" or "muscular action"; or whether it is a predisposing or an exciting cause; or whether, if the latter, it has acted quickly or slowly; and (3) because whether the hernia is one "with a sac" or "interstital" (without a "sac") is not often made sufficiently clear.

Suggestions: Therefore, in describing a case of "traumatic hernia," there should be brought out distinctly, according to its application, one or more of the following facts as to "traumatism" as an etiological factor:

1. *External violence*, as a blow or crushing force, may be an exciting (*i. e.*, direct) or predisposing cause.

When acting as an *exciting* cause it always acts quickly, that is, the resulting " hernia " appears suddenly. Such a " hernia " may be one of two kinds: (*a*) With a " sac," *but* the sac was *already present* (see below, under " muscular action "). In other words, " external violence " as an exciting cause of " hernia " *never* produces the " sac," but only the " contents." (*b*) The other kind is " free interstitial hernia " (hernia without a " sac " and with no " attachment " of parietal peritoneum, already explained).

A case of the author's is illustrative:

> A woman, with no previous history of hernia, was kicked in the abdomen by a horse. A large swelling immediately appeared, with the signs of strangulated umbilical hernia, shock, etc. Operation showed a large coil of small intestine immediately under the skin and fascia, the parietal peritoneum being torn equally with the overlying structures.
>
> Several inches of the gut required excision, followed by end to end anastomosis. Recovery from the operation; but death within thirty-six hours, autopsy showing satisfactory local conditions.

When acting as a *predisposing* cause, " external violence " weakens the abdominal wall, the hernia appearing after an interval and after its immediate exciting cause (see below). In this connection, " external violence " would comprise in addition to a blow or crushing force, a healed wound of the abdominal wall, *i. e.*, the resultant cicatrix.

2. *Muscular action* (" internal violence ") may also be " direct " (or " exciting ") or " predisposing "; and, if the former, it may act " quickly " or " slowly."

When it acts " directly " and quickly, *i. e.*, when the " hernia " appears suddenly, it (the hernia) may be one of two kinds:

(*a*) A " free interstitial hernia "; extremely rare and thought of only as a remote possibility (see case reported in a previous paragraph).

(*b*) The usual kind, that is the hernia has a sac; *but* the sac *has already been formed*. Muscular action, acting " quickly and directly " produces the " *contents* " *only* of this hernia, and never has anything to do with the formation of the *sac*. Therefore, such a hernia, coming on quickly after definite muscular action, if called traumatic, is "traumatic" as regards the viscus only; and this point should be clearly brought out in the description (see also above, under "external violence").

When muscular action acts "directly" but slowly, *i. e.*, when the hernia appears gradually, it (the hernia) may also be one of two kinds: (*a*) The usual kind: There are present both a "contents" and a "sac," which are being, or have been, formed simultaneously. Such a hernia is not often called "traumatic"; but if so, it means usually that traumatism was a *predisposing* cause in the form of external violence, and either as a blow (or crushing force) or as a cicatrix. In this "hernia," "traumatic" should apply to both "contents" and "sac."

(*b*) An "attached interstitial hernia"; quite uncommon; cæcum or bladder or sigmoid (already explained).

When acting as a *predisposing cause,* " muscular action" is well marked and long continued. It serves merely to weaken the abdominal wall; and thus "predisposes" to either "external violence," or to its own further effect, as an exciting cause.

As to the **causation of the "sac,"** in abdominal hernia: As has been shown, the "sac" of an abdominal hernia never has as an exciting or "direct" cause, either "external violence"; or muscular action "acting quickly"; although these causes may *predispose* to its formation.

The only "direct" or "exciting" cause of a "sac" which is formed *after birth* is, muscular action "acting slowly." Therefore the causes of its presence in herniæ which appear suddenly and for the first time, after "external violence" or muscular action "acting quickly," must be prenatal. It should be customary to call all such "sacs" "congenital"; and herein lies another reason for confusion in description, viz.: the calling of many herniæ "congenital" when only the *sac* is such, the "contents" not having entered the sac, it may be, for years after birth.

Applying these considerations to "inguinal hernia," of which the anatomical varieties are much more numerous than those of hernia in any other region, because of developmental variations in connection with the original testicular peritoneal pouch, we should call "congenital" *except the one wherein both "contents" and "sac" are formed slowly and simultaneously,* all these herniæ which possess a "sac," even the one termed "funicular," the anatomy of which is exactly like the regular "acquired" form just mentioned; but it should be made clear just when the term "congenital" applies to the "sac" only; and when to "sac" and "contents."

In **conclusion,** we sum up as follows the different kinds of what are called "traumatic herniæ":

(1) "Free interstitial hernia"; of sudden appearance; rare; always due to "traumatism" as exciting or direct cause: usually external violence; possibly muscular action, acting "quickly."

(2) Hernia with a "sac"; of sudden appearance; direct cause, external violence or muscular action; "traumatic" as regards "contents" only; "sac" having already been formed, *i. e.*, "congenital."

(3) Hernia with a "sac"; of gradual appearance; usually called "traumatic" only when "external violence" (as a blow or crushing force, or cicatrix following a wound of the abdominal wall) is well established as a *predisposing cause;* "traumatic" should then apply to both "contents" and "sac."

(4) "Attached interstitial hernia"; quite rare; of gradual appearance; usually called "traumatic," etc., exactly as just described for No. (3); "traumatic" would then apply both to the organ and to its after-coming "tube" of parietal peritoneum.

Or, to summarize as to *time of appearance:*

A. Herniæ of sudden appearance are: (1) "Free interstitial," rare; or (2) with a "sac" of previous formation; the *sudden appearance* applying to "contents" only.

B. Herniæ of slow or gradual appearance: (1) "Attached interstitial," quite rare; or (2) with a "sac," of which the formation is simultaneous with that of the contents"; direct cause of both forms, "muscular action" acting slowly (a form of traumatism); nevertheless, called "traumatic" only when no doubt exists of "external violence" as a *predisposing cause.*

A CASE OF TRAUMATIC RUPTURE OF THE LIVER.

By John Douglas, M. D.

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A. M.; age, 6; a schoolboy; admitted to Ward I., Bellevue Hospital, June 18, 1903. He was run over by a truck, between six and seven o'clock in the evening. It was impossible to find out anything about the weight of the truck, or over what part of the abdomen it had passed. The patient was immediately brought to the hospital in an ambulance and seen by me at about eight o'clock. Physical examination—Patient in profound shock, in a semi-stuporous condition. When aroused complains of thirst. Skin pale, cold and moist. Mucous membrane blanched. Pupils dilated. Pulse small and weak, but only 116. (The pulse had been more rapid at time of admission.) Temperature 99.2 per rectum. Respirations rapid and shallow. There were several contusions and abrasions on the chin, legs and right lumbar region, and over the left costal arch. The abdomen was not distended, but was slightly rigid and tender. The rigidity seemed equal on both sides, but the tenderness was more marked on the right side. There was some dullness in both flanks, which appeared to change very little on movement of the patient. There seemed to be a slight loss of liver dullness, especially toward the median line, but this was not marked. The patient had been catheterized and the urine found normal.

Believing the case to be one of rupture of an abdominal viscus, with marked internal hemorrhage, laparotomy was immediately decided on, and as the exact location of the traumatism could not be determined, the fact that there seemed to be no point where tenderness was most marked, that there was some loss of liver dullness and that rupture of the intestine was more frequent than that of the liver or spleen, the bladder and kidneys being excluded by the urine examination, the abdomen was opened in the middle line below the umbilicus. On opening the peritoneum, about sixteen ounces of fluid blood escaped and a large quantity of blood clot was found between the loops of intestine. After sponging these out the intestines and mesentery were carefully examined, although no feces were found in the abdominal cavity and no gas escaped on opening the peritoneum. No rent being found and the kidneys and spleen being palpated and found normal, the incision was carried upward almost to the ensiform and the liver examined. The right lobe was normal. The left lobe, from the transverse fissure and falciform ligament, was almost completely severed from the rest of the liver, being only connected with it by a small vein. This portion was hanging in the left hypochondrium by the left lateral ligament. There was also considerable crushing of the anterior part of the quadrate lobe. There was considerable oozing from the raw surfaces, but not very much free hemorrhage, the patient having been greatly exsanguinated. There was nothing to do but remove the severed portion, after ligating the left lateral ligament and the vein connecting the left lobe with the right, and pack the raw surfaces as tight as possible with sterile gauze. The abdominal wound was closed as rapidly as possible with through and through silkworm gut sutures, the packing being led out at the upper angle of the wound.

The patient was in such profound shock that, during the operation, as soon as the bleeding was controlled, an intravenous infusion of 850 c. c. of Billroth's solution was given. On leaving the table his pulse was 160 and very small and weak. The respirations were 40. His condition improved somewhat and at 12.30 A. M. he was out of æther, and at 2 o'clock his shock was much less, but the temperature was 102.4. He was freely stimulated with atropine, caffeine and strychnine by hyperdermic and whiskey in saline enemata. At 7 A. M.—Temperature, 103.6; pulse, 166 and weak; respiration, 28. During the morning his mental condition was bright and he seemed comfortable up to 11.30 A. M., when there was a decided change for the worse and he became delirious, the pulse and respiration being more rapid and weaker. The dressing was examined and, although it was soaked with bile, the hemorrhage had ceased and there were no signs of infection. He gradually became worse and there was some twitching of the hands and of the neck muscles. He had received one-tenth grain of strychnine in divided doses during the seventeen hours since his admission and, as the smallest recorded fatal single dose for an adult is one-quarter grain, it hardly seems possible that these symptoms could have been due to strychnine poisoning, unless the profound hemorrhage and disturbance of the portal circulation contributed to the result. At three o'clock he was again infused, but did not respond to treatment and died at 5.30 P. M., twenty hours after the operation, death being due, I believe, to the result of hemorrhage and shock.

First, in regard to the indications for operation and the diagnosis in this case. The history and symptoms of hemorrhage, accompanied by abdominal tenderness and rigidity and dullness in the flanks, all pointed to rupture of an abdominal viscus, indicating more than shock and called for operative interference. Trendelenberg(1) says that the principal indications for operation in suspected injury to an abdominal viscus are dullness in the flanks and abdominal rigidity, these symptoms being of great value to differentiate from shock. In cases of hemorrhage rigidity appears almost at once and increases during the first hour. It is only in the most severe cases that it fails; and it disappears one or two hours before death.

As to making a positive diagnosis of the viscus ruptured, I do not believe it to have been possible in this case. Eisendrath(2) calls attention to the fact that only a small proportion of the cases of rupture of abdominal viscera show any superficial bruise, and with the exception of cases of rupture of the intestine (from a kick or a blow), it is not of great importance, as the site of the rupture may be on the opposite side of the abdomen from that where the traumatism occurred. He quotes Edler, who reports 184 out of 362 cases of liver rupture without external signs. In the case also, there was an absence of those signs pointed out by Wilms(3) as diagnostic of liver rupture, *i. e.*, pain elicited by pressure over the lower ribs on the right side, and the fact that dullness and abdominal rigidity are more marked on the right side. The absence of these symptoms may be accounted for in this case by the fact that the site of the lesion was the left lobe, which is unusual, as in 83 cases tabulated by Edler the left lobe was only involved twelve times. For the same reason, probably, and also because of the shock from which the patient suffered, there was no pain referred to the right shoulder, a symptom often found in cases of liver rupture.

Concerning the liver dullness, which was diminished but not lost in this case, and which led me to expect possible rupture of the intestine, it was, of course, accounted for by the severed and displaced left lobe. Absence of liver dullness, to which attention was first called by Flint in 1882, as a symptom of free gas in the peritoneal cavity, although a valuable symptom of ruptured stomach or intestine, does not now have the importance attached to it as was previously the case. Robson and Moynihan, in their "Diseases of the Stomach," go so far as to say: "When perforation has occurred, liver dullness is then generally absent, but its presence or absence is void of any significance, and is unreliable as an aid to diagnosis."

It seems worth while to consider what can be done by operation in these cases. Previous to the first operation by Willette in 1888 the mortality of liver rupture, according to Edler, was 85.7 per cent. These statistics disregarded complications however, and Edler(4) found in 96 uncomplicated cases a mortality of 78 per cent. The first operative statistics were those of Terrier and Auvray(5), who in eleven cases gave a mortality of 45.4 per cent. Frankel(6), in 1901, collected 30 cases with a mortality of 43.3 per cent. To Frankel's cases Eisendrath added 7, with a total mortality of 40.5 per cent. To these 37 cases I may add 12 more, including my own, but, unfortunately, without improving the statistics. These cases are reported by Brewer(7) (two), Roesner(8) (two), Fuchsig(9) (three), Finkelstein(10) (two), Blake(11), Wiggin(12), and myself, each one (see reference), and of the twelve cases reported only two recovered. Therefore, of the 49 cases reported there were 25 deaths, making the mortality 52.2 per cent.

In all of these series of cases, however, are included cases complicated by rupture of other viscera, and although the severe nature of the injury necessary to cause rupture of the liver would make these complications frequent, it hardly seems fair to include these cases in determining the mortality of liver rupture. If, therefore, we exclude 5 of Eisendrath's series, three of which had a ruptured kidney, one a ruptured spleen and one a ruptured duodenum, as well as 3 of the 12 cases which I have added; Blake's case having a ruptured spleen and one of Roesner's a ruptured spleen and stomach, the other a rupture of both stomach and duodenum, we have left 41 *uncomplicated* cases, of which 17 died and 24 recovered. A mortality of 41.4 per cent., as compared with the 78 per cent. mortality of Edler's uncomplicated cases without operation.

Obviously these statistics are much more favorable than those without operation, and it is equally obvious that operation gives the greatest hope of saving life in many of the cases, but the question arises, do these statistics give a fair estimate of the mortality of liver rupture? On looking through the reports of cases, one usually finds when single cases are reported, that the operation results in recovery. Hence the report of the case. On the other hand, fatal cases are less apt to be reported, except where to illustrate some other point, a number of cases of abdominal injuries including liver rupture are reported, or where a series of cases are given; and when one looks through such reports the statistics do not appear by any means so favorable (see Brewer, Blake, Wilms, Roesner), although many of these cases are those with severe complications. Therefore, while we must put down as a result of a study of the reported cases, a percentage of *recovery* of 58.6 per cent. in uncomplicated cases and 47.8 per cent. in complicated cases, I believe that the true mortality is probably considerably higher than the figures show, and can only be determined by the report of more of the fatal cases.

Choice of treatment of the liver wound lies between suture and packing as a means of stopping hemorrhage, although the cautery has been recommended, and Hollander(13) and Schneider(14) recommend hot vaporization as a hæmostatic. Schlatter(15), Beck(16), Hahn(17), Terrier and Auvray recommend suture. When a liver wound is sutured, heavy cat gut on a round needle should be used, and great care taken to completely close the deep part of the wound. The objections to suture are the difficulty of accomplishment in some cases, the difficulty of making the sutures hold and not cut through the liver tissue, and of obliterating the space at the depth of the wound. The last two difficulties might, it appears, be overcome by means of the suture devised by Coffey(18), the principal of which is a Y-shaped or X-shaped suture, depending on whether the fissure is incomplete or not, the tension being placed at the crossing of the strands, and on the surface of the liver being supported by a matting of finer cat gut (for further particulars see reference). Beck(19) has also suggested and made use of decalcified bone buttons to relieve tension and tearing out of the stitches after the removal of neoplasms from the liver.

Although Da Costa(20) says that it is necessary to suture the liver to the abdominal wall before packing, as otherwise the packing will only push the liver away, this hardly seems to be borne out by the experience of others. In a large proportion of the cases reported the wound was treated by tamponade, as was my case, and the packing always controlled the hemorrhage. In two cases, however, there was secondary hemorrhage on removing the gauze. The worst bleeding always occurs from the large branches of the portal vein which are pulled out of the liver tissue, and pressure of a moderate degree usually controls the bleeding, the blood pressure not being high. Wounds of the lower always bleed more freely than those of the upper surface of the liver.

It would, therefore, seem that with the exception of those cases in which the liver wound is small and easily sutured, with certainty of obliterating all the space at the depth of the wound, careful, tight packing furnishes the safest and most effectual means of treatment for cases of traumatic rupture of the liver.

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AN UNUSUAL CASE OF NEUROPATHIC ARTHROPATHIES.

By John Douglas, M. D.,

NEW YORK,

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The patient was a German, aged 50, a butcher by occupation. He was admitted to Ward II., Bellevue Hospital, March 17, 1904.

Family History.—Negative.

Previous History.—Was never sick until the beginning of present illness. Does not remember having usual diseases of childhood. Eats regularly and well. Is a moderate drinker, taking an occasional whiskey, including one before breakfast. Is a moderate pipe smoker. Denies any venereal history and shows no lesions of syphilis.

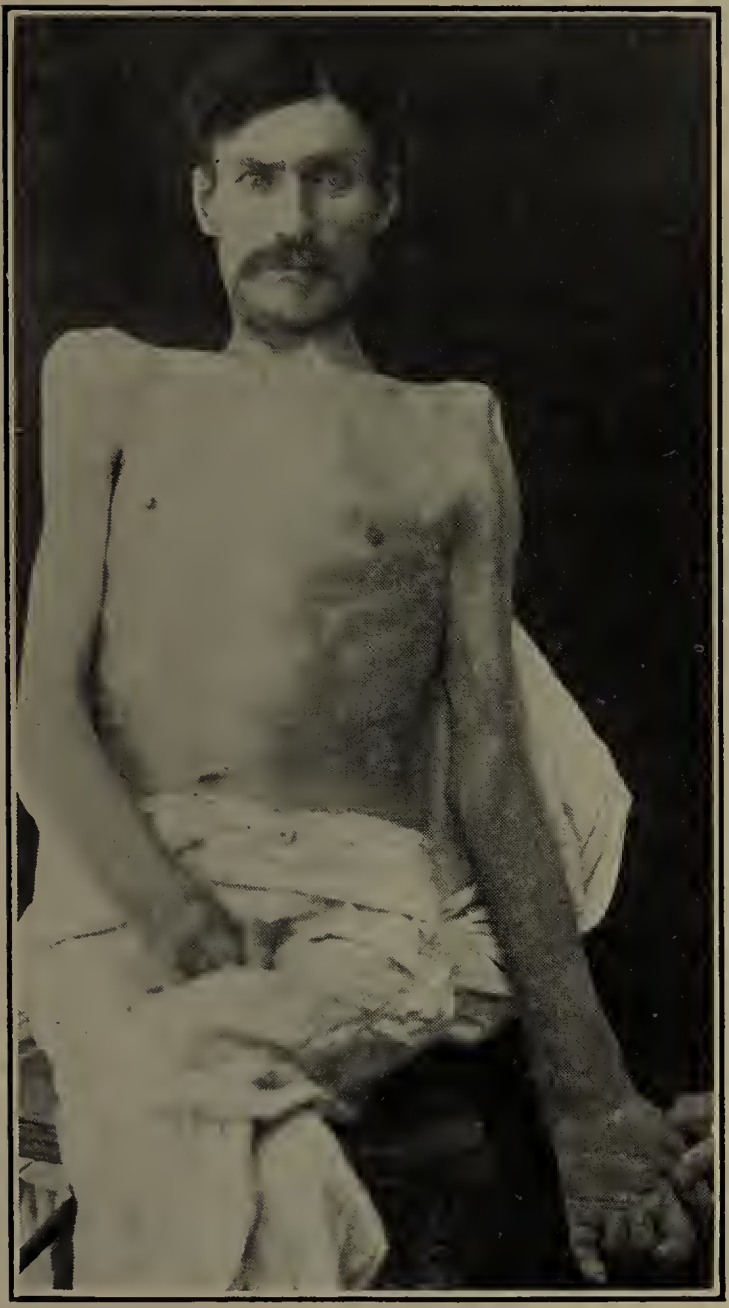
Present Illness.—The first symptom of his disease appears to have been a cellulitis of the middle finger of the right hand, which occurred nine years ago. Developing from a scratch, which was *painless,* and therefore neglected, it resulted in an amputation of the second and third phalanges. He became aware at this time of an analgesia of both hands and arms. Eight years ago, without knowing of any traumatism and without suffering any pain, his left shoulder became swollen and he lost the strength and ability to use the joint as formerly. The swelling slowly subsided and left the joint in its present condition. The following year he had a painless cellulitis of the fourth finger of the right hand, resulting in amputation of the last two phalanges. Five years ago he lost the terminal phalanx of the fifth finger and the following year the last two phalanges of the index finger in a similar manner. He was able to continue his work as before, except for his crippled hand and weak shoulder, and enjoyed good general health up to eight weeks ago. At this time his weight was about 175 pounds. He gave no history of lightning pains or of crises of any kind.

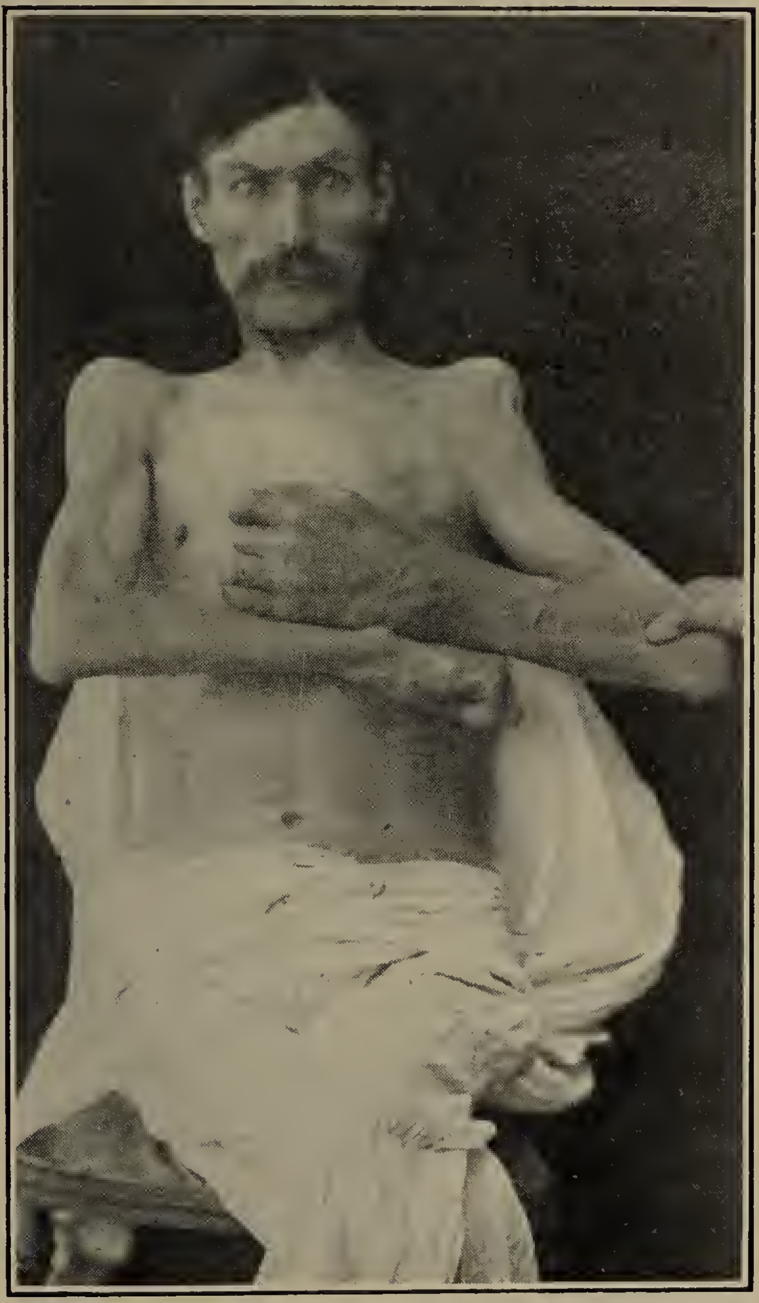
Eight weeks ago patient began to notice a painless swelling and disability in the left wrist and the joints of the fingers of the left hand became stiff and swollen. At the same time the ball of the left foot became swollen. He had some pain in the foot, but was able to walk about on it for four weeks, when a brownish fluid was discharged through the sole of the foot just back of the great toe. He then went to bed and his wife poulticed the foot, which subsequently opened in two other places. At the same time that the joint symptoms appeared the patient began to have a severe cough, which resulted in the development of a right inguinal hernia. He rapidly lost flesh and strength, and began to lose control of his bladder and rectum, which finally became complete incontinence, both urine and feces being voided without his knowledge. It was not until then, three days before his admission, that he consulted a physician, who advised his entering the hospital.

Physical Examination—Patient is of large frame, poorly nourished, weighing about 135 pounds. Heart normal, pulse 108. Arteries show moderate sclerosis. At the apices of both lungs are dullness, bronchial breathing, and voice, crepitant and subcrepitant rales. There is a small reducible right inguinal hernia. The right hand shows the deformity resulting from the amputations described in the history. There is a subcoracoid dislocation of the left humerus, with a complete disintegration of the shoulder joint, the bone being abnormally movable in all directions. The head of the humerus has apparently atrophied and disappeared, together with most of the tuberosities. The glenoid cavity has lost its shape and character. There can be felt an exostosis about two and one-half c. m. in diameter, firmly attached to the axillary border of the scapula, about 3 c. m. below the glenoid, and a smaller bony deposit apparently in the capsule of the joint. There is no swelling or effusion in or about the joint. The muscles are markedly atrophied.

The left wrist is markedly swollen, measuring 2 c. m. larger in circumference than the right, the swelling being more marked on the flexor aspect. It is slightly warm and red, but not tender.

There is no spasm or crepitus, and considerable laxity of the ligaments is present. The small joints of the fingers are swollen and stiff, the fingers being flexed. There is a large gangrenous perforating ulcer on the sole of the left foot, over the head of the first metatarsal bone, surrounded by an area of cellulitis, in which are two sinuses, one over the head of the second, the other over the head of the fourth metatarsal bones. Through the large ulcer projects the black necrotic head of the first metatarsal bone. There is a large pressure sore over the sacrum and several smaller ones are developing over the bony prominences.





From a neurological standpoint, examination shows pupils react to accommodation, not to light; movements of arms very ataxic; patella reflex very much diminished; tongue tremulous; analgesia of both hands and arms to shoulder. No anæsthesia or loss of thermal sense, here or elsewhere, except that patient seemed more sensitive to cold than heat, as is usual in tabes. Muscles showed no reaction of degeneration. Rhomberg's Symptom, or the amount of ataxia in the gait, could not be determined on account of the weak state of the patient and the condition of his foot.

Examination of the urine was negative.

Subsequent History.—The patient rapidly lost strength and died from exhaustion one week after entering the hospital. Unfortunately, an autopsy was refused.

Although neuropathic arthopathies are common lesions, occurring in from 3 to 10 per cent. of all cases of tabes dorsalis, their frequency being differently stated by various authors (3 to 4 per cent., Leszynsky; 5 per cent., Dana; 10 per cent., Charcot), and being even more common in syringomyelia (10 per cent., Sokoloff; 40 per cent., Schlesinger), this case seems to me to be of sufficient interest to report, as it differs in many respects from the typical case of either disease, while the presence of certain symptoms makes the differential diagnosis between tabes and syringomyelia a difficult one.

In regard to the joints which have been affected, their condition is more or less typical of the lesions of either disease. The shoulder shows the atrophic, disorganized, dislocated condition which is usually the terminal result of a neuropathic arthropathy in a ball and socket joint. The upper end of the humerus has atrophied, so as to have the "drum-stick" appearance spoken of by Charcot. The right wrist and finger joints show the typical painless enlargement, although the lesion has existed in these joints too short a time to expect any hypertrophy. The perforating ulcer of the foot is almost painless, although the whole of the head of first metatarsal is necrotic and projects from the ulcer. The other metatorso-phalangeal joints have also undergone disintegration, the sinuses in the sole of the foot leading down to them. According to Sokoloff, the amount of effusion in the joints in cases of syringomyelia is not apt to be as large as in tabetic joints, but this rule seems to be hardly upheld by the facts and may be disregarded as a means of diagnosis. Borchard in a recent article has pointed out that in syringomyelia there is more apt to be an involvement of the surrounding tissues than in the case of tabes, *i. e.*, an oedema of the skin and subcutaneous tissues, together with a muscular atrophy, which is central in origin. There was no oedema in this case and the atrophy present could be explained by disuse. This point then, would favor the diagnosis of tabes.

On the other hand, the distribution of the lesions would point to syringomyelia. As a rule, tabetic arthropathies when multiple are symmetrical, and the lower extremities are affected in a large majority of cases (Chipault, 77 per cent.; Weizsacker, 82 per cent.), the knee and the hip being most frequently involved. In syringomyelia the joints of the upper extremities are most frequently the site of the lesion (Schlesinger, 80 per cent.; Graff, 77 per cent.), arthropathies occurring in the shoulder, elbow and wrist in the order given.

The history of the case is also one of syringomyelia, rather than of tabes. The gradually developing progressive, painless cellulitis of the fingers, with the loss of one or more joints of the affected members, together with the involvement of one of the larger joints of the upper extremity and the analgesia of the hands and arms, is a very common history of syringomyelia. On the other hand, in this case the analgesia was symmetrical and unaccompained by loss of temperature sense, while in syringomyelia the analgesia is usually irregularly distributed or of the " hemiplegic " type, and is usually accompanied by a loss or at least a disturbance of thermal sense.

The patient gave no history of gastric or other crises or of having suffered from ataxia, although this last symptom was demonstrated by examination. Dana and Graf both say, however, that neuropathic arthropathies occur in the preataxic stage of tabes; and Brissaud has called attention to the fact that it is in the sensory form of the disease, where there may be little or no ataxia, that arthropathies are most apt to occur. As opposed to this, Starr states that trophic disturbances are more apt to occur in the stage of ataxia. It must furthermore be remembered that ataxia may occur in syringomyelia.

The age at which the first symptoms appeared in this case, forty-one, favor tabes, the average age at which this disease appears being forty, while in cases of syringomyelia, if the history be carefully gone into, the first symptoms are usually found to date from adolescence, or within a few years afterward. The absence of scoliosis is also slightly in favor of tabes, as it is not an unusual symptom in syringomyelia. Also in the latter, although incontinence of urine may occur late in the disease, loss of control over the rectum is rare.

The two diseases have so many symptoms in common that it is sometimes necessary to leave it to the strongly characteristic symptoms to decide the diagnosis. Thus we have balanced in this case a series of symptoms, with the weight of evidence, if anything, in favor of syringomyelia. There remain, however, two symptoms, the Argyll-Robertson pupil and the loss of knee jerks, which are characteristic of tabes, the knee jerks being usually increased in syringomyelia, and therefore, unless we believe both diseases to have been present, the diagnosis of tabes dorsalis must be made.

It is to be regretted that movement on the part of the patient prevented the skiagraphs which were taken from being of any value, but the accompanying photographs show fairly well the deformities of the upper extremities.

FRACTURE OF THE BASE OF THE SKULL.

By Frederic S. Dennis, M. D., F. R. C. S.,

NEW YORK CITY.

W. D., æt. seven, was admitted to Bellevue Hospital November 13, 1903, with the following history: On the day of admission he was struck on the head by a bale of hay weighing over 200 pounds. The bale of hay fell from a wagon and struck him the blow which produced unconsciousness. He was brought into the hospital by ambulance, and at the time of his admission was suffering additionally from shock. An examination of his injury revealed the presence of a contusion of the scalp, beneath which was an hæmatoma. There was present also a subconjunctival ecchymosis and extravasation of blood about the left eye and side of the nose. There was hemorrhage from the nose and pharynx. The usual examination of the functions of the cranial nerves was made. The patient was treated in the manner suggested by the writer, the attending surgeon, whose routine practice will be presently described. Before discussing the treatment, however, a few remarks will be made in reference to the general subject of fracture of the base of the skull.

Remarks.—Fractures of the base of the skull, in the majority of cases, appear in the form of a fissure, which begins at the vertex and extends to the base. In 146 post-mortem examinations in fractures of the base of the skull reported by Phelps, 133 had their origin in a fissure from the vertex, while only 13 were confined to the base itself; thus it is evident that in a very large proportion of the cases of fracture of the base the injury is a continuation of a fissure from the vertex. Wyman made 34 autopsies in cases of fracture of the skull and found 26 fractures of the base. Thus it also becomes evident that nearly 80 per cent. of the cases of fracture of the skull are fractures of the base. Phelps estimates the frequency of fractures of the skull in general about 60 per cent. Fractures of the base occurred in 89 2-3 per cent. of the 146 cases of fatal fractures of the skull collected by

Dwight, who also reports only 3 cases in the entire number that were not extensions from the vault. In the 131 cases, 78 were limited to one fossa, 10 involved the anterior, 54 the middle fossa, and 14 the posterior fossa. Dwight thinks the most common fracture of the skull is that of the parietal bone and extending in or across the middle meningeal groove through the middle fossa, ending in foramen rotundum or ovale.

The writer, from a personal experience in hundreds of fractures of the skull, finds these proportions in general are in accordance with his own observations. As a rule, a fracture of the vertex is caused either as a result of a pistol shot wound or by a direct localized injury, as a blow upon the skull with a hammer; but, in the majority of cases, any severe violence inflicted upon the vertex usually produces a fracture of the base of the skull. The fissured fracture of the vertex of the skull may extend into the anterior, middle or posterior fossa of the base. The peculiar phenomena attending a fracture into each of the three named fossæ will be considered, after which a study in general of the manifestations which are common to a fracture in any of the three fossæ of the skull. In Dwight's 146 autopsies, 7 involved only the anterior fossa, 50 involved only the middle fossa, and 13 involved only the posterior fossa.

Fractures of the base of the skull into the anterior fossa are generally the result of direct violence, as in the case of a pistol shot wound, or by a blow upon the forehead, or a fall upon the face, or by a thrust into the orbit, mouth, or nose of some instrument or weapon, such as a sword, umbrella or cane. Fracture of the anterior fossa is occasionally caused by a force which drives the inferior maxilla forcibly backward through the glenoid cavity.

Dwight made 146 autopsies in fracture of the base of the skull, and 43 cases involved the anterior fossa.

In these 43 cases, 20 cases, or 46½ per cent., had espistaxis; 4 cases, or 9 1-3 per cent., had hemorrhage from the mouth; 4 cases, or 9 1-3 per cent., had subconjunctival hemorrhage.

Among the signs and symptoms characteristic of fracture of the anterior fossa may be anosmia, immobility of eyeball, blindness, circumscribed anæsthesia, espistaxis, orbital ecchymosis, atrophy optic nerve, optic neuritis, hemorrhage from mouth, hematemesis, paralysis of the third and sixth cranial nerves.

Fracture of the base of the skull involving the posterior fossa is usually the result of a fall backward upon occiput.

It is a very serious injury, since nearly 50 per cent. of the cases are fatal when the fracture involves the fossa, as against 25 per cent. in the other fossæ.

Among the signs and symptoms of fracture of the posterior fossa of the skull may be mentioned: Hemorrhage from mouth or pharynx; temporary glyscosuria, ecchymosis in mastoid and occipital regions, contusion upon back of head, double optic neuritis, epeleptiform convulsions, confusion of intellect, vomiting, headache, coughing of frothy mucous with inability to expectorate it, embarrassed respirations.

If the fracture of the base in the posterior fossa has produced a distinctive lesion involving the glosso-pharyngeal, there will be present aguesia, limited to the posterior one-third of the tongue, and dryness of the fauces and pharynx. If the fracture of the base in the posterior fossa has produced only an irritative lesion there will be present paræsthesia of the pharynx, fauces and tonsils, and a loss of the sense of taste in the posterior one-third of the tongue; also paraguesia and hypersecretion of saliva. If the fracture of the base in the posterior fossa has produced a destructive lesion, involving the pneumogastric nerve, there will be arrest of the heart's action in diastole. If the fracture of the base in the posterior fossa has produced only an irritative lesion there will be present cephalalgia, nausea, vomiting, coughing, hiccough and diminution in the frequency of the pulse.

In the majority of cases the fissure from the vertex of the skull produced by direct violence runs into the middle fossa and extends through the petrous portion of the temporal bone involving the internal auditory meatus. Mr. Hewitt has shown that of 64 fractures of the base, 53 belonged to the middle fossa.

The reason why the middle fossa is the most frequent seat of basic fracture is because the middle part of the vertex is most exposed to injury, and a fissured fracture upon the side of the vertex naturally runs down to the middle fossa, producing a fracture either in the long axis of the petrous bone, or in the region of the sphenoid bone. In Dwight's 146 autopsies, 107 cases involved the middle fossa; 40 cases, or 37 2-5 per cent., had hemorrhage from one or both ears. In 39 cases in which the middle fossa was not involved, 4, or 10¼ per cent., had bleeding from one or both ears. In 33 cases, or 30 9-10 per cent., cases where a fracture of the middle fossa was found, there was no hemorrhage from the ear. Fracture of the middle fossa is usually the result of a fall from a height or occasionally the result of being knocked down or run over by a vehicle.

Among the signs and symptoms characteristic of fracture of the middle fossa may be: (1) Hemorrhage from external auditory meatus; (2) escape of cerebral tissue from external auditory meatus; (3) paralysis of third cranial nerve and chorda tympani; (4) ecchymosis mastoid region; (5) contusion over parietal or frontal-parietal region; (6) facial paralysis, with or without deafness.

If deafness is present immediately upon receipt of accident it is likely to become permanent; if later, the prognosis as regards hearing is more favorable. In 106 cases of fracture of the base collected by Bidwell, 15 of these cases had facial paralysis.

The mechanism of fractures of the base has been a subject about which much controversy has arisen. Von Bergmann, Bruns and others have given the subject much study, and believe that these fractures of the base depend upon certain elastic properties which the skull possesses. Sancerotte thought that the vibrations of the bony wall of the skull travelled according to certain laws of physics and accumulated at points directly opposite and caused the fracture. Aran believed that fractures of the base are fractures by eradiation, while Félecet believed that fractures of the base of the skull were caused by force propagated along weaker arches, and that in these arches, and not at the buttress, a solution of the continuity occurred.

The shape of the cranium explains the mechanism of fracture. Félecet has demonstrated that the skull is formed of pillars or buttresses of thickened bone between which the bone is thinner.

These buttresses pass from the base to the vertex in the median line. Félecet held that fractures were usually found to involve the bony surfaces between the buttresses.

Mechanism of fractures of the base of the skull, like those of the vertex, are likewise explained by certain laws of physics. In studying the mechanism of fractures it must be remembered that there are three fossæ on either side of the median line of the skull. The skull is not one sphenoid, but is made up of at least three sphenoids, if not six in all. There is an occipital and frontal pole, also a vertex and base pole of the skull. Any force applied, for example, to the occipital pole, will not only dilate both frontal poles, but also the anterior part of the middle fossæ—*i. e.,* the cavity of the greater wing of the sphenoid and the anterior wall of the posterior fossa. This theory, formulated by Schultze, explains, according to his idea, the single and multiple laceration of the brain from contre coup.

Wahe and Körber believe that fractures of the base travel in the direction of the violence, or, in other words, parallel to the direction of the pressure. Messerer demonstrated that the skull caved in, so to speak, at the sight of greatest pressure, or else pulled apart at the point where the skull is most stretched. In other words again, a fracture of the base may be due to compression, in which case the fracture runs at right angle to the axis of pressure, or to a bursting, in which the line of fracture runs parallel to it.

The theory of a bursting fracture of the skull deserves careful study. The bursting fracture is one transverse to the line of action—*i. e.*, the line of action of the force is assumed to be in the axis of the sphenoid. This axis is shortened by the action of the force, and, consequently, the equator is increased and the fracture occurs transverse just the same way as if each meridian was separate, and owing to the fact that the ends are approached the bone would fracture at its mid-point.

The tearing fracture of the skull is one parallel with the line of action of the force; in other words, a fracture more or less in the meridian of the sphenoid. The fracture is explained by the fact that, with the increase of the equator by the action of the force in approaching one pole to the other, the parallels of latitude are also increased; those nearer the equator are relatively more increased than those nearer the poles. By the increase of the parallels of latitude it is fair to assume that in certain parts the bone cannot accommodate itself to this distention, and it is consequently torn more or less in a plane that is parallel with the line of force, viz., more or less in a meridian.

Schultze believes that, in a fracture of the base of the skull, the force which acts upon the skull changes the shape of the skull, and that whenever bone accommodates itself to the change of shape readily by its elasticity, no fracture will occur; whenever, however, the bone is too rigid to accommodate itself to the change of shape, the bone will fracture.

In reference to the remote consequences after fracture of the skull, Bullard has traced them in 70 cases, and his conclusions are that in 37 cases there were no apparent symptoms, and in

7 cases there were present serious symptoms, but in 4 of the 7 it was doubtful whether the serious symptoms were due to the fracture of the skull. Headache, deafness, dizziness and inability to resist the action of alcohol were the most frequent symptoms found.

In regard to the treatment of fracture of the base of the skull, it may be said that operative interference is rarely called for in pure fractures of the base. It occasionally happens that the orbital plate of the ethmoid, or the petrous portion of the temporal bone, are fractured, in which case surgical intervention is required to remove the loose fragments, and also to prepare the wound for aseptic healing. Fractures of the base associated with fractures of the vault of course require operative interference. The writer has adopted the rule in all cases of serious head injury in which unconsciousness is present, to incise the scalp and examine for fracture. Recently the writer has done this for diagnostic purposes, and in both cases a fracture was present and cerebral substance escaped from beneath the scalp. In neither of these cases was the scalp injured. In one case the man died in ten hours from shock of the injury, and in the other case the incision revealed the presence of a fracture and the intracranial pressure was relieved caused by hemorrhage and loose cerebral matter and bone, and the patient recovered

Incision in the scalp saved this patient, though there was no visible evidence of fracture present. It occasionally happens that a blood clot may be so large that it threatens life by pressure. In these cases the trephine may be used in the same way as in the vertex. This can be done when the clot is in the anterior fossa, by placing the trephine just above the superciliary ridge. This same procedure can be done when a clot is in the posterior fossa by placing the instrument upon the bone near the insertion of the trapezius muscle after its fibres of insertion have been divided.

Fracture of the base of the skull has been one of those injuries which the surgeon has treated purely upon the expectant plan, and with the conviction that death is sure to follow. This view of the lesion has been entertained for years by hospital surgeons, and there is certainly a consensus of opinion among surgical writers in regard to the prognosis. All the older standard text books and monographs are in accord in reference to the mortality. During the past few years the writer's attention has been especially directed to a study of these cases of basilar fracture, and he will endeavor to demonstrate here that fractures of the base of the skull are amenable to surgical treatment. The writer will also endeavor to point out the clinical fact that the great majority of these cases die from causes which can be wholly prevented by the surgeon. There has been but little encouragement to the surgeon to attempt any treatment in these cases of basilar fracture, when one reads in the latest edition of a large standard work on surgery "that any treatment directed to the lesion itself is out of the question."

Fractures of the base of the skull should be considered in the same category as compound fractures of the vertex of the skull.

Fractures of the base of the skull possess the same essential characteristics as other compound fractures; they should therefore be treated by the same exact principles of aseptic surgery.

Some surgeons may dissent from the first proposition, viz., that a basilar fracture should be considered in the same light as a vertex fracture. It is evident, however, that every compound fracture has air admitted to the seat of the fracture, and that this is the case in fractures of the base of the skull. If the anterior fossa is the seat of lesion, air gains entrance to the fracture through the nasal cavities. If the middle and posterior fossæ are involved, air gains access either by the external auditory meatus or by the Eustachian tube through the pharynx and nasal cavity. The entrance of air to the seat of fracture, which is a *sine qua non* for the existence of a compound fracture, is not the only point that basilar fractures possess in common with all other compound fractures. The basilar fracture is associated with hemorrhage, laceration of nerves, venous thrombosis, septic phlebitis, fat embolism, cerebro-spinal shock, pyæmia, septicæmia and abscesses, and, in fact, with all the complications that arise in connection with other compound fractures. Even the cerebral lesion is not confined to basilar fractures, because it occurs in fractures of the vertex. It is thus evident that fractures of the base do not differ in any essential particular. The view may be even advanced that the cerebral lesions in vertex fractures may be more severe than in basilar fractures, because in the latter the cerebro-spinal fluid in a measure protects the brain from concussion, and thus diminishes the shock.

The treatment of compound fractures in general is simple. Every patient suffering from fracture of the base, if the immediate shock is not too profound, should be removed to the operating-room. Every outside preparation having been made to make the toilet aseptic, the head should be held firmly and the entire scalp shaved, after which it should be thoroughly washed with soap and water, and then irrigated with a 1 to 500 bichloride of mercury solution. Attention should next be directed to the irrigation of the external auditory canal and the nasal cavities. Both of these should be rendered thoroughly aseptic, after which the ears should be packed with iodoform or bichloride gauze, and some absorbent cotton plugs placed in the nose. The air can filter through cotton, but micro-organisms cannot pass through it. In irrigating the nasal fossæ the surgeon must exercise great care lest the fluid enter the stomach and produce toxic effects. There remains now only one other possible channel of infection to the seat of fracture, and that is the Eustachian tube. This may carry sepsis to the interior of the cranial cavity in a fracture of the base. The arrangement of the ciliæ in the tube is such as to propel air and mucus *toward* the nasal cavity; but during the act of deglutition the current may be reversed, and infection thus reach the base of the brain. In the ordinary form of fracture of the base, where the membrana tympani is ruptured, the discharges make their exit through the auditory canal. In the exceptional cases where the membrane is not ruptured, the serum, blood and cerebro-spinal fluid find their way into the nasal cavity and the pharynx, and thus these cases are most exposed to sepsis. If the unruptured membrana tympani is examined in a basilar fracture, the membrane will be seen puffed out on account of the presence of fluid behind it, and the membrane will have lost its natural lustre. These are the circumstances under which it may be wise, under an anæsthetic, to puncture the membrana tympani and thus allow the blood and the cerebro-spinal fluid to escape through the external auditory canal. The diversion of the discharges from the Eustachian tube into the ear makes it possible to keep the fluids permanently aseptic. Having now rendered the head and all the avenues of approach to the intracranial cavity surgically clean, it remains to apply a dressing that will keep these parts permanently free from any source of infection. This object is best accomplished by the use of a cap made of loose bichloride gauze or carbolized wool, over which absorbent cotton and a bandage are applied. The cap should completely surround the entire head, cover in the ears, and protect every channel leading down to and communicating with the seat of fracture. The cerebral viscera are thus protected from the entrance of septic germs. This same dressing should be made from time to time during the repair of the fracture, and under the same strict and rigid rules of antiseptic surgery.

The reasons for dressing fractures of the base of the skull with a scrupulous attention to the minutest detail in antiseptic surgery are at once obvious. If a patient survives the immediate shock of the fracture and lives twenty-four hours, he is almost certain to die from causes which can be directly attributed to sepsis. Among these causes may be mentioned basilar meningitis, cerebral abscess, pyæmia, suppuration in the petrous portion of the temporal bone, septic phlebitis, encephalitis, leptomeningitis and other complications due to infection.

If the surrounding parts and the discharges are kept aseptic then sepsis cannot occur, and this removes at once all the causes of death due to septo-pyæmia. The writer does not pretend to state that it is impossible for a patient with fracture of the base to die except from a cause due to sepsis, but to state that nearly all these cases terminate fatally from causes which are due to sepsis. A reference to statistics will demonstrate that deaths occurring in fractures of the base of the skull, independent of shock or sepsis, are extremely rare, and that nearly all these cases die, therefore, from causes which can be prevented by the surgeon. The writer would go even further, and recommend that all cases of head injury, with hemorrhage from the ear or from the nose, be treated with antiseptic precautions. There are many cases where a fracture of the base of the skull did not exist, but where a hemorrhage was present from the ear or the nose, and in the course of ten days a fatal meningitis developed. Every practical surgeon can recall such cases as these, where the meningitis has its origin in septic contamination of the hemorrhagic or other discharges. The old and good rule in surgery, "Never to look lightly on any injury of the head, however trifling it may appear," is one that, if strictly observed, may be a means of saving life where otherwise that life might be sacrificed. During the past few years the writer has seen a large number of cases of basilar fractures. Most of these cases died from shock within twenty-four hours, but recently six cases recovered under the plan of treatment just described. By placing basilar fractures in the same category as compound fractures, and treating them as such, there is a prospect of recovery in cases which have hitherto been considered fatal. If fractures of the base can be saved, it is another triumph of antiseptic surgery.

NOTES UPON EXPERIMENTAL OBSTETRICS.

By J. Clifton Edgar, M. D.,
Visiting Obstetrician to the Emergency Hospital of Bellevue Hospital.

I.—The Effect of Different Postures of the Pregnant Woman Upon the Antero-posterior Diameter of the Pelvic Inlet and Outlet.

The True Conjugate.—The true conjugate can be estimated from the diagonal conjugate by constructing a triangle formed by the two conjugates and the symphysis pubis, of which the diagonal conjugate corresponds nearly to the hypothenuse and the true conjugate to the base. The diagonal conjugate, the known quantity, is the longest of the three sides, and the true conjugate, the unknown quantity, can be obtained from it by subtracting on an average 1 inch (2.5 cm.). The amount to be deducted, however, will vary with the height, thickness and

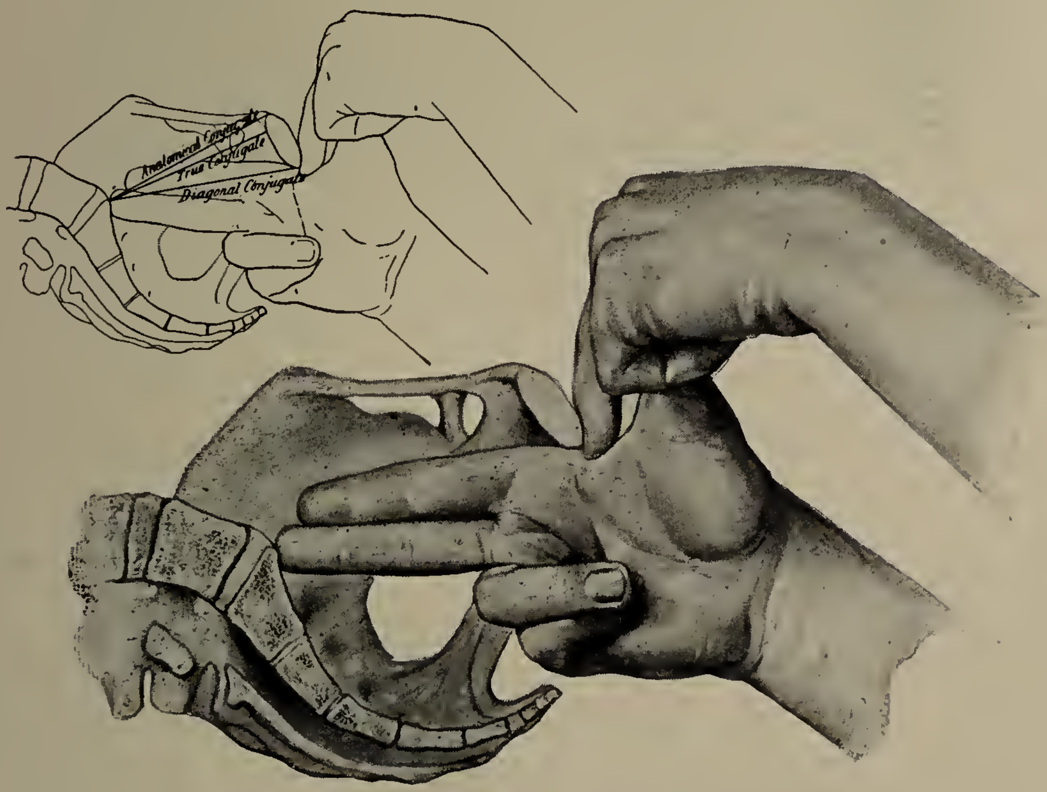


FIG. I.—DIGITAL METHOD OF MEASURING THE DIAGONAL CONJUGATE OF THE PELVIC INLET. (FROM A PHOTOGRAPH.)

inclination of the symphysis and the height of the sacral promontory. When the symphysis is 1½ inches (3.75 cm.), or over, ¾ inch (1.905 cm.) should be subtracted from the diagonal conjugate; and when it is less than 1½ inches (3.75 cm.) a little less is to be subtracted. The estimation of the conjugata vera by this plan can only be approximated, since it depends upon so many variable quantities.

By this method it is very common for marked variations in the length of the diagonal conjugate to be found in the same patient by different observers, and in the estimate of the true conjugate we have a further source of inaccuracy (Fig. 1).

Exact pelvimetry demands that the true conjugate shall be measured directly by means of some metal non-flexible instrument that shall eliminate the foregoing sources of error.

Such an instrument is that of Farabeuf.

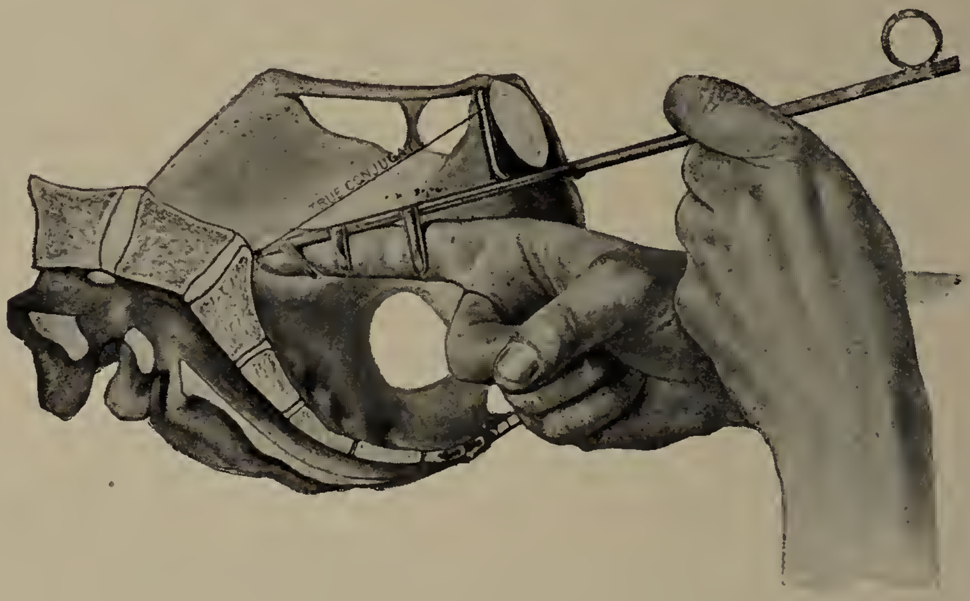


FIG. 2.—DIRECT INSTRUMENTAL METHOD OF MEASURING THE TRUE CONJUGATE OF THE PELVIC INLET WITH THE FARABEUF PELVIMETER. (FROM A PHOTOGRAPH.)

True Conjugate with the Farabeuf Pelvimeter.—This resource was introduced into pelvimetry by Farabeuf for the purpose of lengthening the index finger, in case the accoucheur should be unable to reach the promontory, either by reason of the shortness of his finger, or because of unusual dimensions of the pelvis (Fig. 2). While fingers of average length are sufficient for pelvimetry in contracted pelves, this is by no means necessarily the case in general pelvimetry, and, therefore, the device of Farabeuf is excellent in routine and obstetrical practice. The custom of reinforcing the finger with a vesical sound is open to criticism, from the fact that it may be given a wrong direction;

and a further disadvantage is the absence of a device to indicate the exact position of the symphysis. The thimble-like pelvimeter consists of two delicate steel arms, which are parallel for a short distance, but which then diverge and afteward roughly assume the contour of a tapering forefinger. This frame is provided on its inferior surface with two incomplete rings, designed to fit the first and second phalanges of the exploring finger. The elasticity of the steel arms permits the rings to slip over a finger of any normal dimensions. Attached near the extremity of this steel frame is a delicate horseshoe-shaped piece of steel, which turns in either direction, up or down, and which constitutes the extension to the exploring finger (Fig. 2). The parallel portion of the steel arms also constitutes a groove, along which slides the measuring rod, which is bent at its terminal end into a right angle. This bent portion is intended to enter the urethra in order to abut against the internal aspect of the symphysis. The proximal end is provided with a ring, while the upper surface of the rod has a graduated index (Fig. 2). With this pelvimeter the obstetrical conjugate can be measured directly. The steel arms are introduced against the promontory, followed by the passing of the measuring rod into the bladder (Fig. 2).

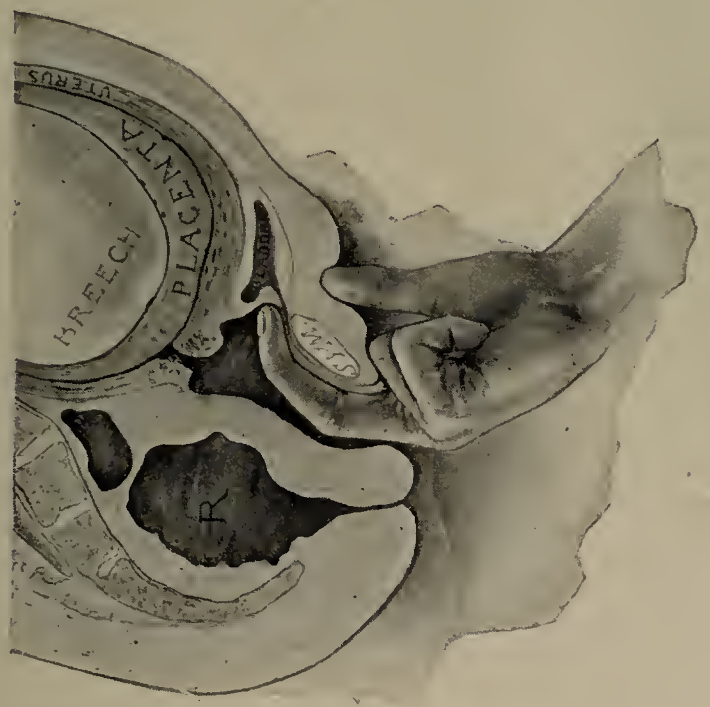


FIG. 3.—MEASURING THE HEIGHT AND THICKNESS OF THE SYMPHISIS WITH THE FINGERS.

Since 1895, whenever opportunity offered, I have been making observations at the Emergency Hospital upon both primiparal and multiparal to determine this point. The ages of patients ranged from 18 to 40 years.

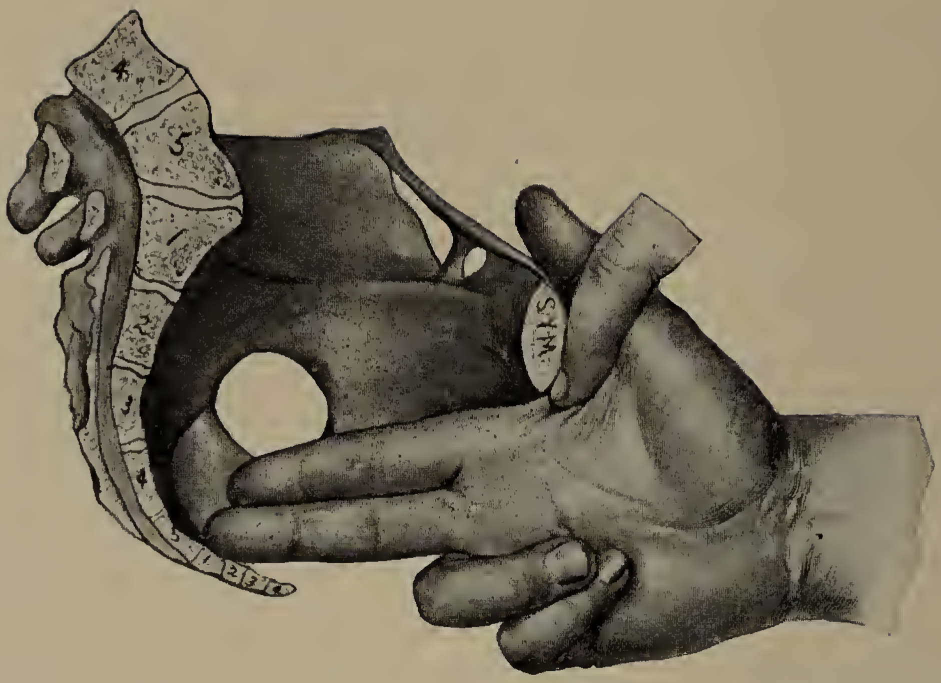


FIG. 4.—DIGITAL METHOD OF MEASURING THE ANTERO-POSTERIOR DIAMETER OF THE PELVIC OUTLET. (FROM A PHOTOGRAPH.)

EXPERIMENTS.

1. The true conjugate was measured with a Farabeuf pelvimeter through the bladder with patients in the dorsal posture and the thighs moderately flexed upon the abdomen.
2. The same measurement was made as in 1, but with the patients' thighs strongly flexed against the abdomen.
3. The true conjugate was measured with a Farabeuf pelvimeter through the bladder with patient in the Walcher posture.
4. The antero-posterior diameter of the pelvic outlet was measured digitally with patients in the dorsal posture and the thighs moderately flexed upon the abdomen.
5. The measurement was made as in 4, but with the thighs strongly flexed upon the abdomen.
6. The antero-posterior diameter of the pelvic outlet was measured digitally with patients in the Walcher posture.

CONCLUSIONS.

1. In the Walcher posture the true conjugate was increased in every case, the variations being from one-fourth to half an inch (0.635 to 1.27 cm.).

I found the greatest gain in muliparæ near full term and under thirty-five years of age.

The smallest gain was in primiparæ near full term and over 30 years of age.

2. The antero-posterior diameter of the pelvic outlet was diminished in all cases by the Walcher posture from ¾ to 1 cm.

3. Moderate flexion of the thighs upon the abdomen does not, in my experience, affect the length of the true conjugate.

4. Strong flexion of the thighs against the abdomen causes a decided lessening of the true conjugate.

5. Moderate flexion of the thighs upon the abdomen does not affect the length of the antero-posterior diameter of the pelvic outlet.

6. Strong flexion of the thighs against the abdomen causes an appreciable increase in the antero-posterior diameter of the pelvic outlet.

7. The difference between the antero-posterior diameter of the pelvic inlet in the dorsal posture as compared with that of the Walcher will depend upon whether the measurement of the former is taken with the thighs moderately or strongly flexed against the abdomen.

The greatest differences I found when the thighs in the dorsal posture were strongly flexed.

In 1898, at the New York Maternity Hospital, I measured several series of cases from among the pregnant waiting women with a Farabeuf pelvimeter in the lithotomy posture with both moderately and strongly flexed thighs, and also in the Walcher posture, and the results do not differ materially from the above.

II.—Cleidotomy—A Neglected Operation.

Cleidotomy, a division of both clavicles in dead fetuses as a preliminary to delivery of the shoulders, has for its object the diminution of the bisacromial diameter of the fetus.

This simple operation, rarely if ever mentioned in obstetric text-books, has never, in my opinion, taken its proper place in obstetric surgery, as a valuable means of lessening maternal morbidity and mortality.

How often do we witness a difficult extraction of the shoulders in a generally contracted inlet or outlet after the perforation and extraction of the head.

I have seen an hour consumed in the delivery of the shoulders. I have seen the head dragged from the body by the excessive traction.

During the past ten years cases have been admitted to my service at the Emergency Hospital with the head delivered and the shoulders still within the pelvis. These last, however, are usually midwife cases.

As a matter of routine in these cases, I always divide the fetal clavicles, and it is amazing how the diminution of the bisacromial diameter thus produced renders the subsequent extraction of the fetal shoulders a comparatively easy task, and quickly and completely changes the clinical picture for the better.

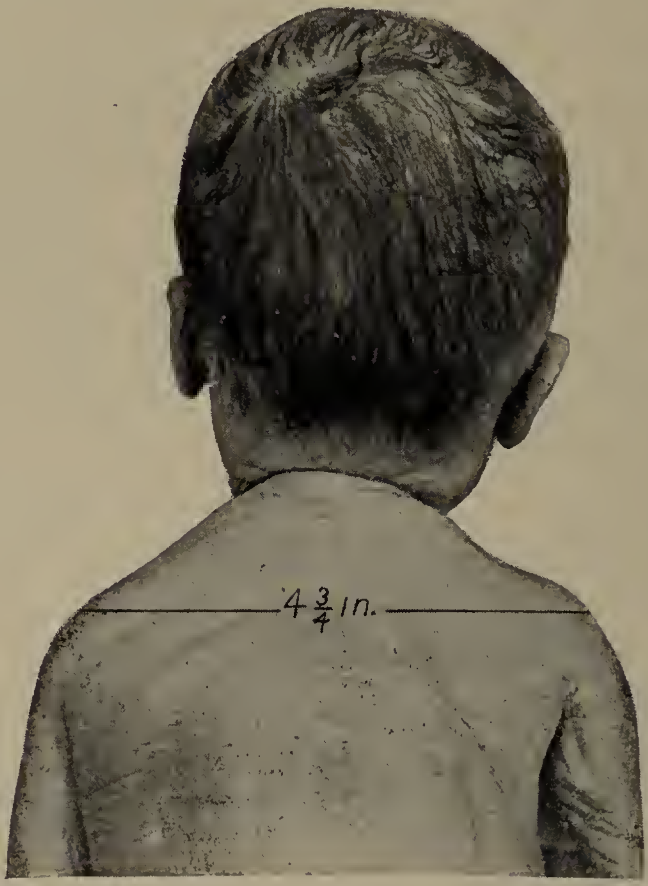


FIG. 6.—FETAL CADAVER BEFORE CLEIDOTOMY.

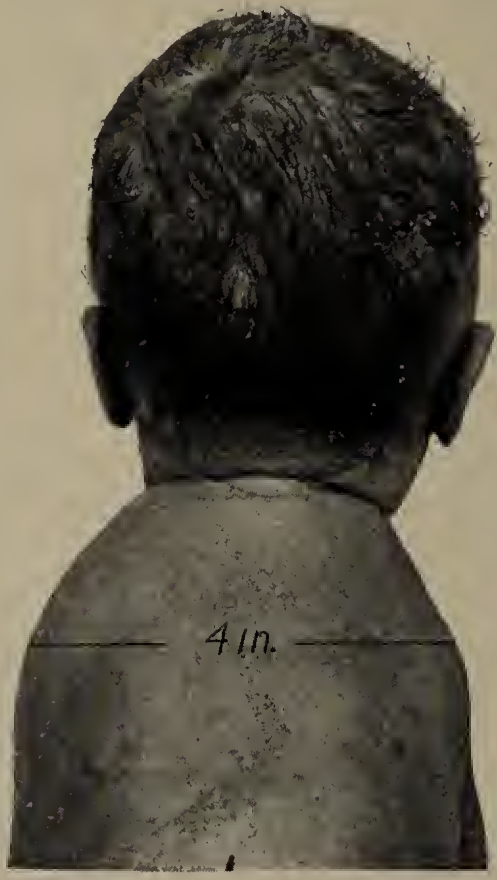


FIG. 7.—FETAL CADAVER AFTER CLEIDOTOMY.

I have resorted to the operation repeatedly at the Emergency Hospital, in cases in which the birth of the head had been accomplished by forceps or perforation and cranio-traction, and the shoulders resisted all ordinary methods of extraction.

The operation has its limitations. I have never been able to reduce the bisacromial diameter more than two inches in full-term fetuses. From the measurements I have made at the bedside and from experiments upon six fetal cadavers I find that the reduction in the bisacromial diameter produced by the operating ranges from ¾ of an inch to 2 inches (Figs. 6 and 7).

Operation.—For the operation I always use a pair of heavy straight or curved obstetric scissors of the Dubois type, two fingers of one hand being used to guide the blunt points to the middle of each clavicle (Fig. 5).

It is usually necessary to strongly extend or flex laterally the fetal head so as to give room for the use of the scissors (Fig. 5).

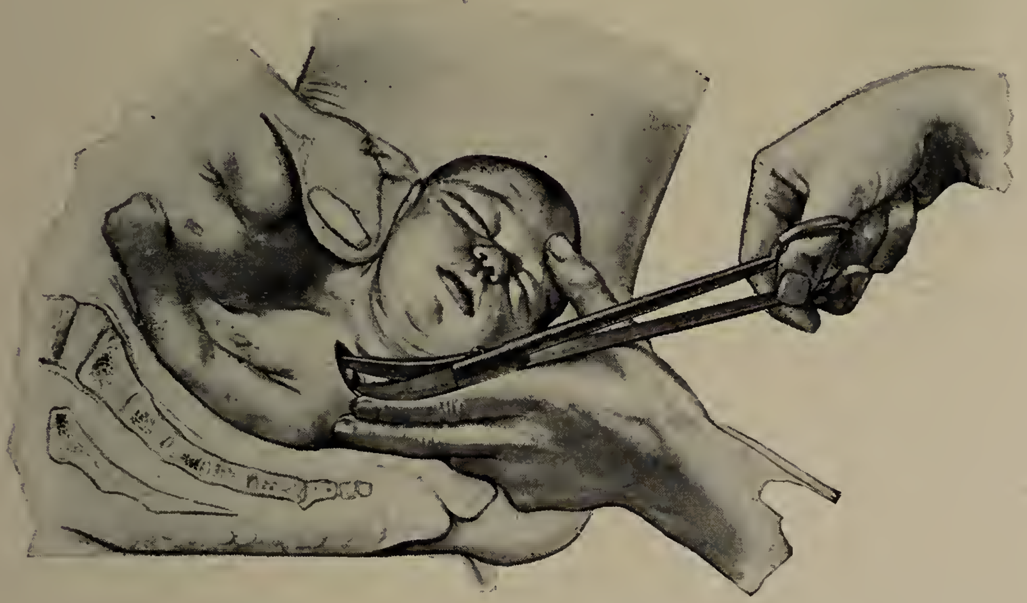


FIG. 5.—OPERATION OF CLEIDOTOMY, PERFORMED WITH LONG CURVED SCISSORS.

III.—EXPERIMENTAL THERAPEUTICS OF THE PUERPERIUM.

What place have drugs and various non-medicinal agents in hastening the termination of the puerperium as regards lochia and involution and in the prevention of subinvolution?

I have experimented with various drugs and non-medicinal means at the Emergency and New York Maternity Hospitals during the past ten years, and at the Emergency Hospital these experiments were limited to the use of ergot.

The following special printed history blank was used for the recording of the results, and the records were entered upon the chart by the resident head nurse, but all measurements of the depth of the uterine cavity were made by myself with a blunt-pointed notched uterine sound on the tenth day of puerperium.

The following is the form of the history blank:

CASE No. ....

Name .... Age .... Para .... Birthplace ....

Presentation .... Position .... Month of gestation ....

Labor *spontaneous or not, duration, complications, full-term or not, cervical, vaginal, and perineal lacerations,* ....

PUERPERIUM.

1. Height of uterus above symphysis:
   1st day .... 2d .... 3d .... 4th .... 5th .... 6th .... 7th .... 8th ....
   9th .... 10th .... 11th .... 12th .... 13th .... 14th .... 15th ....
2. Posture in bed *dorsal, dorsal—elevated, lateral, abdominal, was posture frequently changed?* ....
3. After-pains *occurred on what days? duration, intensity, treatment,* ....
4. How were bladder and rectum emptied? *catheter, bed-pan, kneeling in bed, commode or vessel out of bed,* ....
5. What day first sat up in and left bed? ....
6. Was lactation normal? ....
7. Medication *drugs, frequency,* ....
8. Douches *vaginal—frequency, intra-uterine—frequency,* ....
9. Lochia *less than usual, normal quantity, profuse, excessive, ceased on what day?* ....

EXAMINATION ON DISCHARGE.

DAY OF PUERPERIUM ....

1. Quantity and character of lochia present *bloody, rusty, white, none,* ....
2. Position, sensitiveness, and mobility of uterus? ....
3. Dilatation at internal os *closed, admits index readily, index passed with difficulty,* ....
4. Depth of uterine cavity from external os? ....
5. Did sound cause any bleeding? ....
6. Old and fresh cervical, vaginal and perineal lacerations? *location, extent, character,* ....

Remarks ....

Signed ....

Address ....

Date 189

At the Emergency Hospital I have experimented only with fluid extract of ergot, given in drachm doses t.i.d., to 9 primiparæ and 11 multiparæ.

Observations were also made upon 18 primiparæ and 14 multiparæ to whom practically no medication was administered, with the exceptions of two drachms of ergot at the completion of the third stage.

The observations appear to show that in the cases in which ergot was given throughout the puerperium, that on the tenth day the lochia was less than in the series in which no medication was used; also that the internal os closed earlier and the depth of the uterine cavity was slightly less.

Although the figures appear to indicate the above, still I am inclined to look upon the results as *entirely negative.*

It is interesting to note, however, that the continued use of ergot during ten days of the puerperium did not interfere with breast-feeding.

I may perhaps be permitted to state here, that since the above experiments were made at the Emergency Hospital I have experimented more extensively at my service at the New York Maternity.

	No Medication during Puerperium (Ergot ʒ ii End Third Stage) 18 Primiparae.	No Medication during Puerperium (Ergot ʒ ii End Third Stage). 14 Multiparae.	Ergot throughout Puerperium. (Ergot ʒ i t. i. d). 9 Primiparae.	Ergot throughout Puerperium. (Ergot ʒ i t. i. d). 11 Multiparae.
Average duration of labor	16 hours 44 minutes	10 hours 38 minutes	14 hours 37 minutes	16 hours 1 minute.
Average height of fundus—				
Average height first day	6.67 inches	6.88 inches	6.50 inches	6.68 inches.
" fifth day	4.29 "	4.43 "	5.00 "	4.15 "
" tenth day				
After pains—				
Absent	100 per cent		100 per cent	
Present		75 per cent		70 per cent.
Posture in bed	Frequent change	Frequent change	Frequent change	Frequent change.
Maternal nursing	100 per cent	100 per cent	100 per cent	100 per cent.
Artificial feeding				
Lochia in puerperium—				
Natural	83.33 per cent	75 per cent	100 per cent	50 per cent.
Scant	10.66 "	12.50 per cent		37.50 per cent.
Profuse		12.50 "		12.50 "
Vaginal douches	End third stage	End third stage	End third stage	End third stage.
Medication	Ergot ʒ ii end of third stage.	Ergot ʒ ii end of third stage.	Ergot ʒ i t. i. d.	Ergot ʒ i t. i. d.
		Examination on Tenth Day.		
Lochia—				
Moderate				44.44 per cent.
Scant	53.33 per cent	80 per cent		11.11 "
Absent	13.33 "	20 "	88.88 per cent	22.22 "
Profuse	33.33 "		11.11 "	22.22 "
Dilatation of internal os—				
Admits index	80 per cent	81.81 per cent	55.55 per cent	83.33 per cent.
Closed	20 "	18.19 "	44.44 "	16.16 "
Average depth of uterus	3.66 inches	3.56 inches	2.97 inches	3.55 inches.
Bleeding caused by sound—				
None	6 cases	6 cases	5 cases	2 cases.
Slight	9 cases	5 "	3 "	6 "
Moderate			1 case	1 case.
Sensitiveness of uterus—				
Absent	75 per cent	33.33 per cent	100 per cent	75 per cent.
Moderate	25 "	66.33 "		25 "

These latter experiments included the administration of quinine throughout the puerperium to 22 primiparæ and to 23 multiparæ; the giving of strychnine for ten to twenty days before and ten after labor to 30 multiparæ; and the administrating of strychnine throughout the puerperium to 48 primiparæ and 54 multiparæ.

I will only say at this time my results here, as at the Emergency Hospital, I consider negative with the exception of those obtained in the use of strychnia.

OBSERVATIONS ON SOME OF THE MOLECULAR CHANGES CAUSED BY LIGHT.

By Charles A. Leale, M. D.

Light has always possessed many mysterious properties since the creation, when God said, "Let there be light." Modern science has done much to reveal the hidden actions of light, yet much more remains to be developed to demonstrate the wonderful power and influence of light over the numerous diseases affecting both the vegetable and animal creation.

The main source of light to the earth is the sun.

The form of radiant energy known as light is one of the most powerful therapeutic agents placed at the command of the physician. He may artificially produce an intense light, so powerful as to quickly destroy life, or he may shield the individual from most of the rays of light, so as to be in apparent total darkness, or he may divide or separate the rays of light so as to utilize any desired color of the spectrum.

More than twenty-five years ago, while endeavoring to investigate the causes of the troubles in a large number of children whom I saw in convulsions, I came to the conclusion that many were the result of light-stroke. I daily passed hundreds of helpless infants in open baby carriages, their faces in the full glare of the midday sun. Sometimes the children were asleep, oftener they were awake, with their contracted facial muscles plainly revealing their agony. I prepared a paper on the subject and read it before one of our scientific societies, citing instances under my own observation where, from exposure to the direct rays of light, convulsions, meningitis and strabismus followed, and urged all who ever saw one of these helpless victims to properly explain the danger and do everything possible to educate the public on this subject.

DANGER OF ELECTRIC-LIGHT SHOCK.

It is my duty to caution lecturers, clergymen and all others who are compelled to use artificial light at their public reading desks to have proper intervening screens covering the incandescent lamps, to divert the dangerous rays and mollify those necessary for light; especially if at the time physically or nervously depressed, or if they have any degeneration of the heart whereby its action could not quickly respond to any shock.

During the past few years there have been several instances of shock produced by the direct rays of light transmitted through the clear glass globes of the incandescent lamps; especially when a surgeon at a brilliantly illuminated operating table had placed the unshielded back and top of his head in a position to receive the full force of the powerful rays; also after-dinner speakers at banquets, who have been placed at tables directly under and in the line of concentrated rays of intense light, have been known, not only temporarily, but sometimes fatally, to be affected by "light-shock." The dangerous rays of light have passed through the covering of the cranium to the brain cells and instantaneous molecular changes have ensued. The pneumogastric nerve, apparently, is the most quickly affected. The heart muscle fails to contract, the organ becomes engorged and acutely over dilated, the laryngeal muscles are held in firm rigidity, the physiological action of the stomach is checked and the man has his entire circulatory system stopped, his breathing has ceased, his stomach has become acutely distended, he is in a condition of profound electric-light shock, and in a few minutes is either dead or deeply affected until his vital force has overcome the paralysis and he has again control of the functions of life.

DANGEROUS RESULTS OF THE USE OF THE ELECTRIC LIGHT IN OPTHALMOSCOPIC EXAMINATIONS.

Quite recently a young lady of excellent physique and great culture, whose eyes had been in a condition of fatigue, came a long distance to New York City, to consult me in regard to profound nervous phenomena following the prolonged use of the opthalmoscope reflecting the intense rays of the incandescent light directly into her eyes. While under the influence of atropia, the fierce rays at a very close range had for such a long period been used that the patient became exhausted and faint. The illuminations she stated were several times repeated, until her agony had forced a discontinuance of the clinical study of her normal eyes. This young lady had returned to her home so completely changed that fears were entertained in regard to her sanity. Her vision had altered and her mind easily became confused. In this instance the long-continued impinging of the violent rays of light, through largely dilated pupils, had produced inevitable molecular changes in the extremely sensitive neurotic membranes in the interior of the eye, and had probably also extended along the course of the optic nerve to the delicate cells of the brain.

The Roentgen rays, although generally represented by the commonest algebraic symbol for an unknown quantity, are now known to produce under certain conditions such profound molecular changes in animal tissue as may result in any degree of structural alteration, from necrosis to a simple staining.

One of my patients, who frequently used the X-rays and had taken every precaution to avoid injury, had scores of permanent brownish black freckles appear on the callous skin on the palms of his hands, a portion of the skin generally supposed to be immune; demonstrating molecular changes caused by the intense rays of light. These black spots on the palms of the hands very much resemble the skin of the faces of certain admixtures of the brown and the black races of mankind, with a lesser admixture of blood from the white race. In this combination I have often seen on the faces of individuals of these mixed bloods the intensely black freckles in the otherwise yellow black skin of a certain variety of negroes.

The permanent staining of the skin of delicate blondes resulting from the use of preparations of silver, either in powder, ointment or more especially the solutions of nitrate of silver, makes it evident that they should never be used under any circumstances, as there are harmless remedies equally efficient. I have often seen otherwise beautiful faces permanently discolored by the use of silver or bismuth preparations, the broad dark circles under the eyes clearly showing the metallic stain. These discolorations are entirely different from the maculæ resulting from a permanent hyperæmia of the blood vessels of the skin, which are affected by heat and cold, and almost completely disappear under pressure, while the metallic stains of silver remain unchanged when subjected to such tests.

Several years ago an American physician and honorary president of the International Medical Congress, after years of practice in one of the most deadly malarial parts of our country, came to me to care for him during the short remaining period of his existence. For his malarial troubles he had taken enormous quantities of quinine and for the relief of his tuberculous ulcerations of the stomach and bowels he had for years taken Pil. Argenti Nitras. After returning to the East his skin lost some of the color produced by the sun's rays, he became anæmic and was such an object of horror that life became miserable. His skin was permanently changed; he was an Argyria.

If the physician had known the uselessness of Pil. Argenti Nitras to overcome ulcerations of the bowels, if he had for a moment considered the lasting effect of silver staining upon the skin, or if he had known the extreme sensitiveness of silver to the action of light, he told me he would never have resorted to its use.

For many years, on my summer vacations in the Adirondack, Catskill or White mountains, the inland country or at seashore resorts of both Europe and America, I have observed the different effects of light upon the skin on different parts of the same individuals.

On the entire seashore of the United States most blondes, when exposing their skin to the unsheltered rays of the summer sun, have as a result molecular changes both in color and tissue. The black plaques on the back of the hands and on the face, and the thickening of the skin prone to repeated desquamations being quite different from the changes produced in the skin of the same class of individuals either in the mountains or on the shores of our great inland lakes.

Again, when the same individuals enter the Torrid Zone and remain exposed for a few months to the sun's rays there is a diffuse general darkening of the skin, but with the most prolonged stay, after years of exposure out of doors, there remains a sharply defined difference between the skins of the different races of mankind, the Caucasian clearly continuing entirely different from the Negro.

I have often noticed while at the seashore in midsummer the effects of radiated light and heat on matter when exposed to the sun's rays, and how parts of the deeper structure beneath the skin of man were differently affected. For instance, the acutely active skin of the back readily becomes blistered, while that of the anterior portions of the body, arms, legs, chest, abdomen and face are less so; but the skin of the eyelids has a remarkable shielding power to the rays of light, protecting the underlying structure so that it remains almost immune to destructive molecular changes.

In the mountains, remote from the seashore, after weeks of exposure to the sun, wind, dry or moist atmosphere, the skin of the white inhabitant of New York City generally assumes a light bronze color, free from spots.

I have often noticed the gradual effects of age in diminishing the pigment of the skin of negroes, especially those of pure ebony mothers and white fathers. These mulattoes, although living in the South, after they pass the age of sixty years, very rapidly lose the dark color of the skin simultaneously with the same changes which are being produced in the hair, until the age of eighty, when the hair and the skin may closely resemble those of their paternal ancestors at the same age.

I have observed great differences between the action of the concentrated rays of light and those of the concentrated rays of heat upon living animal and vegetable cells.

During the past thirty years there have been many therapeutic experiments on the action of light when rays have been passed through blue glass; also in the use of red window glass in the treatment of smallpox to prevent pitting. The ultra-violet rays have also been very extensively used in the treatment of many diseases. But I believe better results may be attained when all the colors of the spectrum are utilized.

For many years I have used the double convex sun-glass to send concentrated rays of light deep into the base of the structure of epitheliomata, and thereby in superficial cancers have produced permanent cures without any resulting cicatricial scars.

These effects of the power of light, also the wonderful photodynamic influences on the retina of man and the increase of the electrical current produced by light are some marvelous examples of a great energy, which exists in all creation.

Often on a summer day have I sat beneath the shade of some giant maple tree of the forest. There was intense heat in all the surrounding atmosphere, not a breath of air was in apparent motion, the clouds even seemed at rest. Many thousands of leaves above my head were motionless, yet upon close scrutiny, while nature and all vegetation appeared asleep, I have perceived a solitary leaf in constant motion, trembling in the greatest agitation. On closer inspection that leaf was seen to be younger and of a more verdant hue than all the others. Differing from them, it hung as a pendulum, with its tip pointing to the earth and with a motion that can be explained by light acting upon its molecules and generating an electric current which passed from it toward the ground. The action of that beautiful solitary leaf, continuously kept in electrical motion, representing a natural electro-motor, was entirely different from the action of the leaves on the aspen tree (*populus tremulus*), where the mobility depends on the leaves being suspended by leaf stalks flattened laterally, so that by a gentle breeze they are kept in perpetual motion.

I have cited these examples of the direct effects of rays of light in producing molecular changes in animal and vegetable cells to show what may be detected by any careful observer.

Many of the causes of these changes have remained mysterious until the past few years, but science is gradually unfolding the book of mysteries, and accurate observers, scientific thinkers, meditative minds and competent writers are giving to the world results never before supposed possible to be attained.

REPORT OF A CASE OF TORSION OF THE UTERUS.

By John F. Erdmann, M.D.,
Attending Surgeon, Gouverneur Hospital.

Torsion of the uterus is of such rarity that I deem this case the most interesting occurring in a service extending over a period of seven years in this hospital.

A widow, fifty years of age, had passed her menopause five years before. During her early married life she had had three miscarriages, but had never gone on to complete pregnancy. In the month of August, 1903, she had an attack of abdominal pain, accompanied by considerable shock, which lasted three days. This was followed in about a month by another attack, less severe and of about two days' duration. Third attack and the last occurred one week ago; that is, January 2, 1904. When I saw her in consultation on the afternoon of January 10, she had been suffering for two days and a half with intense and excruciating pain in the abdomen. There was considerable collapse evident, a temperature of 102 F., with a pulse of 126. The abdomen was markedly distended, simulating a pregnancy of the ninth month, with a fair degree of fluctuation over the left side. Vaginal examination, no cervix to be palpated within a range of four inches, but a large boggy mass pressing down on the posterior vaginal wall; also a large fluctuating mass pressing down on the anterior wall. Rectal palpation revealed also a boggy mass, rather fixed in its nature. This condition was considered as a large fibroid with a twisted pedicle, or a large ovarian cyst with torsion. The patient was taken to Gouverneur Hospital within the hour, prepared, and within two hours was operated upon, a large median incision being made. Upon cutting through the peritoneum a quantity, not less than two pints, of chocolate colored, thin, flaky fluid was expelled, and a large blue-black body presented in the incision. Upon extending the incision three inches above the umbilicus we found this mass appearing as though it were a large fibroid of the uterus itself; but upon extruding the tumor it proved to be an enlarged fibroid uterus which was attached to a pedicle one and one-half inches long by three-quarters of an inch wide, and twisted two and a half times from right to left. The broad ligaments were also twisted and the tube and ovary on both sides blue-black, markedly oedematous and thickened. Even at this time we were unable to state what the pedicle consisted of. When the mass was untwisted a clamp was placed on the broad ligament on each side. Scissors were rapidly applied and tumor cut away, and then ligatures applied en masse and by sutures to the remains of the broad ligament. At the middle position of the pedicle where no clamp was placed the bladder fundus was found within a half inch of the incision, and also a small part of what proved to be the cervix was found, less than three-quarters of an inch in width laterally and less than one-half inch anterior-posteriorly, with an extremely small opening for the canal. A complete toilet of the peritoneum in the pelvis was made. The chocolate colored fluid remaining was wiped out with dry gauze. The appendix, which was found to be flexed and bound down by adhesions, sclerotic and atrophied, was released and removed. The abdomen was sewed up after having put two liters of salt solution into the peritoneal cavity. A prompt reaction from shock was noticed and for the first forty-eight hours an absolute negative convalescence was observed. The beginning of the third day the patient began to vomit; had some meteorism, and looked as though a sharp attack of septic peritonitis was about to establish itself. All food-stuffs, medications, fluids, etc., were ordered to be kept from the patient, excepting such as could be absorbed by the rectum. A movement of the bowels followed within twelve hours when again the patient showed absolute negative convalescence. From this time on the patient recovered perfectly and was discharged from the hospital in the third week, the wound having united per primam. The uterus, which weighed 7½ pounds upon removal, was found to be a general fibroid mass with one large mass on the left side which had undergone cystic degeneration to such a degree as to cause the sense of fluctuation which was manifested at the time of examination.

GENERAL REPORT OF A SIX WEEKS' SERVICE IN THE ERYSIPELAS PAVILION OF BELLEVUE HOSPITAL.

By J. C. Ayer, M. D.,

Adjunct Assistant Surgeon, Bellevue Hospital; Consulting Surgeon, Nassau County Hospital, etc.

During a service in the Erysipelas Pavilion covering a period of six weeks, from January 1 to February 15, 1904, I had under my care one hundred and twenty-nine cases of various infectious diseases, mostly erysipelas; but for the purposes of this paper I have selected seventy-nine typical cases of erysipelas from which to draw the statistics and deductions which follow.

I have endeavored to trace, as far as possible, the etiology of the cases which came to my notice; to give the average duration of the disease, the average of maximum temperatures, the course and termination, and the percentages of the occurrences of the most important factors in the disease, in order that one may obtain, at a glance, an accurate picture of a typical case.

To Dr. Hamilton, Surgical Interne in charge of the erysipelas wards, I am indebted for the careful manner in which the histories were taken, and for other details which have been of great use to me in preparing this paper.

In drawing up the following table, I have, of course, taken under consideration only such cases of erysipelas concerning the diagnosis of which there could be no doubt:

Number of cases	79
Type—	
Facial	94.6 per cent.
Extremity	5.4 per cent.

Etiology—

Idiopathic	63 per cent.
Wounds	23.7 per cent.
Rhinitis	6.5 per cent.

Other sources of infection:

Varicose ulcers	I
Tertiary lesions of nose	I
Blister from sinapis	I
Ear discharge	I
Furuncle	I

Average duration	9.4 days
Transitory febrile albuminuria present	32.6 per cent.
Average of highest temperatures	104.3

Termination—

By crisis	50.2 per cent.
By lysis	49.8 per cent.

Deaths due to erysipelas	1.25 per cent.
Deaths from complications	13 per cent.

Viz.: Alcoholic cerebral œdema; delirium tremens; pneumonia; uræmia; pyæmia; carcinoma of pylorus; typhus fever (?); chronic nephritis.

As the cases under discussion varied considerably from the classical types of the disease, as described in the literature on the subject, it has seemed to me that a brief sketch of the clinical features of the disease, as seen in the wards, would be of some value.

Invasion—The onset of the disease was manifested in the large majority of cases by a chill, general prostration, nausea and vomiting, sudden rise of temperature—often as high as 105 F.—and the characteristic erythematous patch. This local sign was generally coincident with the constitutional symptoms, but in a considerable number of cases it preceded or followed them by a period of several hours. In three cases there was no history of the initial chill at all, and only slight constitutional symptoms. These cases mostly ran a mild course, with only a slight elevation of temperature. In one, the duration of the disease was much shorter than normal, while the other two showed very little variation in point of time from the average case.

Course—When the patient first comes under observation the temperature is almost invariably high—103 to 104.5F. There is marked prostration, anorexia, and sometimes a persistent nausea.

The local symptoms are marked and slowly progressive, so that, at the end of four or five days, in a facial case, both sides of the face may be involved, the ears hugely swollen and the eyes closed. Extension of the facial lesion is generally backwards to the neck and back, by way of the scalp, and rarely by way of the chin and front of the neck. From the neck the spread is down the back, under the arms to the chest, down over the buttocks and backs of the legs to the heels. The front of the abdomen generally escapes. From the face, extension often takes place as far back as the pharynx, and in two of my cases, pronounced streptococcus cultures were made from this locality. It is remarkable that the symptoms of this form of extension should be so mild, the patients complaining of nothing but a moderate degree of sore throat.

A mild delirium often accompanies the uncomplicated facial cases, but in markedly alcoholic patients it is almost invariably present, and is generally of serious import, often indicating and preceding the onset of a fatal delirium tremens or wet brain.

A moderate leucocytosis is generally present, ranging from 9,000 to 16,000; but in one migratory case the leucocyte count was as high as 30,000.

The albuminuria which was present in 32 per cent. of my cases was usually of a transitory character, sometimes accompanied by casts, and cleared up with the fall of temperature; but on the other hand, those patients who were suffering from severe chronic nephritis at the time they were attacked by erysipelas, invariably died of uræmia.

As will be seen by reference to the table, the great majority of deaths was due to intercurrent diseases and conditions for which the erysipelas was only indirectly responsible; and, moreover, the greater part of the patients suffering from chronic disease of the vascular system, chronic nephritis and chronic alcoholism, easily fell victims to a fatal pneumonia, uræmia, delirium tremens or œdema of the brain, nor did the degree of the acute infectious process always stand in a similar relation to the liability to a fatal complication. A patient having one of the lowest maximum temperatures recorded, viz., 102.8, fell a victim very early in the disease to a fatal attack of wet brain.

In the so-called "migratory" or "wandering" types of the disease, after a period of twenty-four to forty-eight hours, during which the local erythema remained stationary, a new involvement of the skin took place, often accompanied or preceded by a chill, sudden elevation of temperature, nausea and vomiting, the extension advancing in a serpiginous manner at the rate of about two inches or more in twenty-four hours. Fruitless attempts were made to check this advance by painting the skin with a narrow "fence" of pure carbolic acid in solution with glycerine at a distance of half an inch from the advancing margin; by excoriating the skin with a scalpel and painting the wound with the same solution, and by a constricting band of adhesive plaster or of collodion. All these devices were absolutely without effect, the disease advancing across the "dead line" at the same uniform rate as before.

Termination.—The manner of termination of the disease in the cases under consideration was both by crisis and by lysis, the former being slightly more frequent than the latter, though neither sufficiently predominating to justify its being called characteristic.

The fall of temperature and the beginning of desquamation were generally coincident, and at the same time the condition of the patient began to improve. Convalescence was generally rapid and uneventful, excepting in those cases which had the migratory form of the disease, and in which, in consequence, the duration of their illness had been considerably lengthened.

Treatment.—Recognizing the inadequacy of the usual methods employed in the treatment of erysipelas from my previous experience when Interne in the hospital, I wished to make some observations on the effects of the anti-streptococcus serum on the disease, but I was greatly disappointed by the fact that the manufacturers were unable to make deliveries until so near the end of my service that the number of cases treated with the serum was too small to be of much statistical value. The results in the few cases treated were, however, so exceedingly encouraging that I was pretty well convinced of its value; so much so, that if the opportunity should again arise for its employment, I would use it without hesitation to the exclusion of the archaic methods heretofore employed as a routine. Painting with ichthyol and collodion, wet dressings of aluminum acetate or boric acid, while they may have had some slight beneficial effect on the local process, had absolutely no effect on the constitutional symptoms, as for as I could determine; and the internal administration of the various drugs recommended was apparently absolutely without effect.

In one case, where the apparent effect of the serum treatment was most pronounced, the patient, a woman of 28, was admitted on the second day of the disease, with an involvement of the left half of the face, and a temperature of 104.8; she received an injection of ten c. c. of serum into the abdominal muscles on the day of admission. In the next twenty-four hours the temperature fell to 102.5, when she received the second injection. At the end of the third period of twenty-four hours the temperature had again fallen to 99.8, and from that time on it remained down at normal, or nearly so. At no time subsequent to the first injection was there any further extension of the disease, and the patient was only moderately sick. The total duration of her sickness was not over five days, which is much below the average for patients with such a high initial temperature. The disease ended in this case by crisis, while in another case, similarly treated, with an initial temperature of 105, the termination was by lysis and desquamation was established on the sixth day.

Complicated Cases.—In addition to the usual facial types of the disease, the service furnished several cases of a rather unusual character, a description of which may be of interest:

Case I.—G. S. (colored), 23, a pantryman, was admitted to one of the medical wards on January 17, 1904, with the following history: About Christmas the patient became conscious of a pain in his left hand, which gradually extended until it involved both shoulders, both elbows, both wrists and the right knee. On the night before admission he had a chill, nausea and vomiting, and sore throat. Has had a rash for two weeks. On admission, joints were swollen and red. Five days later he was transferred to the Erysipelas Pavilion, where a diagnosis of facial erysipelas was made. Patient complained of pain in knees and ankles, and had a peculiar papular, discrete, hemorrhagic rash on the backs of his arms and hands, and on the backs of both legs, extending from the sacro-iliac joint to the dorsa of the feet. Patient had previously given a history of syphilis two years ago, and on examination, whitish plaques resembling mucous patches were found on the buccal mucous membrane, and the epitrochlear glands were palpable. Pupils, heart, lungs, liver, spleen and other viscera apparently normal. Urine amber, acid, clear, 1,020 specific gravity; trace of albumen; no casts. Leucocyte count, 11,600, divided as follows: Polynuclear, 59 per cent.; large lymphocytes, 14 per cent.; small lymphocytes, 15 per cent. eosinophiles, 2 per cent.

For two and a half days before his transfer to my division, his temperature had ranged from 99 to 100.2, but with the advent of the erysipelas it shot up to 105.2, and remained up in the neighborhood of 102 to 104.5 for twelve days, when he died.

On the third day he complained of headache, vomited several times, and hiccoughed at intervals. This hiccoughing continued to increase until it became constant, day and night. At first it could be stopped temporarily by traction on the tongue, or by deep pressure with the thumbs in the region of the pneumogastric nerves in the neck; but these and all other methods at length failed to relieve the spasm, and it continued uninterruptedly until his death. The findings at the autopsy were only of a negative value. There was a moderate degree of hypostatic congestion of the lungs, and a mottling of the surface of the liver, but no pathological lesion of sufficient importance to account for his death. The tentative diagnosis, for want of a better, was typhus fever. The unusual features in the case were the peculiar rash, which persisted after death; the incessant and uncontrollable hiccough, and the vomiting—all without giving any definite clue to the nature of the malady.

Case II.—Erysipelas of leg and thigh, complicated by pyæmia. A. S., male, 65 years; patient was admitted to the wards on February 5, with a patch of erysipelas on the outer side of the leg below the knee, and another on the thigh just above the knee, which developed subsequently to the first patch. Physical examination showed marked senility, with arterio-sclerosis and general tremors. The erysipelatous patches were moderately superficial, being somewhat boggy on palpation, but not fluctuating. Temperature, 102.8; pulse, 96; respiration, 20. Urine, clear, amber, acid, s. g. 1,018; trace of albumen, no sugar, no casts. Leucocytes, 32,800; hæmaglobin, 90 per cent.; red cells, 2,800,000. Patient gave a history of some injury to the right shoulder one week prior to admission. Temperature ranged at about 103.5 for three days and then dropped from 1 to 2 degrees. The patches of erysipelas had in the meantime been treated by wet aluminium acetate dressings, and had remained about the same.

On the fifth day after admission patient complained of severe pain in the injured shoulder, which, upon examination, was found to be considerably swollen and boggy. He then began to be delirious at night, though the temperature continued to fall until it ranged between 99 and 100.5. He was now in a state of almost continuous delirium, and had frequently to be restrained. He had complete incontinence of urine and feces.

On the night of the seventh day the temperature shot up more than 4 degrees—to 103.8—and he died in twenty-four hours from that time. The autopsy showed a large collection of pus under the deltoid muscle and external to the joint cavity; no other purulent focus was found in the body, with the exception of the lesion below the knee, which, when opened, showed a small pocket of pus comparatively deep down in the tissues. This was evidently a case of pyæmia, the metastasis taking place at the site of the injury about the shoulder—a *puncta minimæ resistentiæ.*

Case III.—J. M., male child, one month old. Erysipelas migrans, complicated by pyæmia. Patient admitted to the ward on January 10, with a facial erysipelas. This rapidly spread over the scalp and down the back, and in four days the whole surface of the body was involved. On the tenth day a small, fluctuating tumor was discovered over the lower lumbar region. The skin over it was not involved, and it had the appearance of a cold abscess. Two days later four more masses of a similar character were discovered, one on each elbow and two on the left thigh. The next day these masses were opened and a thin, yellow pus was evacuated. On the same day a similar swelling appeared over the left wrist joint. Five days later this swelling was also opened and two drams of a similar pus were evacuated. The child died two days after this last operation, and a diagnosis of pyæmia was made. The temperature curve was from the beginning characteristic of pus, and the leucocyte count was 21,800.

THE PREVENTION OF SHOCK DURING SURGICAL OPERATIONS.

By Stephen Smith, M. D.; LL. D.,

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In the early days of the use of anæsthetics in Bellevue Hospital sudden collapse during an operation was a very common and often most disastrous event. In common with other members of the then resident staff I had my proportion of cases of desperate struggling with the imminent symptoms of immediate death. The method of administration of the anæsthetic was: entire avoidance of food during the day of the operation, a liberal dose of whiskey or brandy within fifteen minutes of the use of the anæsthetic and free supply of the ether or chloroform. We used Squibb's ether and it was administered at first under the immediate direction of Dr. Squibb, Senior.

When I became responsible for my own cases for operation, as Visiting Surgeon, I changed Dr. Squibb's method by giving the stimulant an hour before the operation. This was an improvement, for the heart was already fortified against depression, when etherization began, and the distressing vomiting, which seemed to be induced by the stimulant just taken, rarely occurred.

It happened early in my service that a woman entered my wards suffering from cancer of the breast. She was upwards of sixty years old, very fat, with an extremely weak and intermittent pulse. A consultation decided that the removal of the breast was necessary, but that the condition of her heart would require the most careful attention.

While she was waiting for the operation I had occasion to assist Dr. Sayre in an operation for the removal of a cancerous breast, and his patient proved to be the counterpart of my patient. She was about the same age and precisely the same build and fatness, with a weak and intermittent heart. During the operation there was a slight hemorrhage when the heart ceased to beat and a complete collapse followed. Every possible effort was made to restore the action of the heart, but after a long struggle the task was given up and the body was removed from the table to the mortuary.

The result of this operation impressed me as to the dangers of my own case and I delayed the operation several days to determine the best means of protecting my patient. The object to be secured was, evidently, two-fold, viz., first, to strengthen, and, second, to sustain the heart. By the usual method of procedure the patient was deprived of food for twenty-four hours; the stimulant was given just before the operation and was usually followed by vomiting when etherization began; depression of the heart followed with complete failure of this organ on the sudden occurrence of a loss of blood.

It had long been an axiom of military surgeons to operate on the wounded while they are under the influence of stimulants, for it was the universal experience that they bore the accidents of hemorrhage far better than did those who were not stimulated. It was even advised to operate during the heat and excitement of battle for the purpose of taking advantage of the extreme tension of the heart's action, and thus avoiding collapse from the loss of blood.

The plan of procedure which I decided to follow was to feed the patient and stimulate the heart long prior to the operation. A French surgeon had recently advised hot milk as the best heart stimulant before and during an operation, preferring it even to brandy as it not only immediately stimulated the heart but by its nourishment sustained its action for a long period. Acting upon this suggestion I prepared my patient as follows, viz., at 8 o'clock on the morning of the day of the operation she took two ounces of whiskey in a tumbler of milk, so hot that she had to sip it. This was repeated at 10 and 12 o'clock. The operation was at 2 o'clock, at which time the patient had taken three tumblers of hot milk and six ounces of hot whiskey. I found her in a quiet sleep from which she awoke in a dreamy mental state; her pulse was 96, full and regular; her face was flushed; her eyes were suffused; her hands and feet were warm; her temperature was 99.

The operation and dressing required thirty minutes. The features of the operation which attracted special attention were as follows: The very small amount of ether required, estimated at half an ounce; the entire absence of efforts at vomiting; an unvarying pulse of 96, full, strong and regular; a gradual recovery from the anæsthetic without vomiting, but followed by quiet sleep. The interesting incidents of her convalescence were a continuance of the fullness and regularity of her pulse for several days without stimulation; rapid healing of the wound which was complete on the tenth day, though there was a wide dissection; a vigorous appetite from the first; an immediate recovery of even better health than she had previously.

Not long after this operation a young woman entered my wards suffering from a long continued suppuration of the knee-joint surfaces. She was in a state of extreme prostration, with a flickering pulse, death-like pallor, great excitability and constant dread of being disturbed by dressing her knee. She came to the hospital for the express purpose of having her limb amputated, but was terrorized at the bare mention of the operation. On such occasions her pulse would flicker for a moment and then disappear, requiring her head to be lowered, by removing the pillows, to prevent fainting.

Conferring with Dr. George T. Elliott, then on duty on the Medical Division, it was decided to undertake the hot milk and whiskey treatment during the forenoon, and at the hour of the operation he would visit the ward, where he was unknown, in the capacity of a clergyman and induce our patient to inhale chloroform as a remedy for the immediate relief of her suffering, all the preparations for the operation to be in waiting in an adjoining room. The ruse succeeded admirably; the patient took an ounce of whiskey in half a tumbler of hot milk three times at intervals of two hours. Dr. Elliott visited the ward at 1 o'clock, wearing a white cravat, and spoke with each patient in turn until he came to our patient in whose case he took a special interest. He found her well under the influence of the stimulant, face flushed, eyes suffused and pulse quite full, averaging about 100 per minute. She was very communicative about her condition, wished she could have her limb amputated without knowing it and readily consented to smell the cordial which he had in a fancy vial. He poured a few drops upon a handkerchief which he held near her face; the effect was immediate; she was removed to the operating table; the amputation was quickly performed and the patient returned to her bed. The same peculiarities were noticed in this as in the first case, viz., but little of the anæsthetic was required, there was no evidence of a disturbed stomach, the pulse did not vary in frequency and strength after the first excitement of the anæsthetic. Her restoration from the anæsthetic was without vomiting or headache, and it was several days before she became aware that her limb had been amputated. She complained of pain in her toes and some smarting pain above the knee, but was surprised that her former pain had disappeared. One day she noticed a blood spot on the sheet at the knee and was greatly alarmed lest a hemorrhage would occur. She was then informed that her leg had been amputated several days. The good news was followed by the most profuse expressions of gratitude and delight. Her recovery of perfect health was extremely rapid and satisfactory.

From that time forward I have used this simple means of preventing shock during operations and have never seen an accident of that kind when this course of preparation has been pursued. As compared with the use of strychnia and similar heart stimulants, the hot milk and whiskey has this decided advantage, that it affords a quickly digested and absorbed food to an exhausted and depressed system and a mild but long-continued and effective stimulant to the circulatory and digestive organs. The convalescence of feeble persons after operations has, in my experience, on account of this feeding and stimulation, been far more rapid and complete than in cases where the heart stimulants alone, transient in their effects, have been employed.

A CONTROLLABLE ARTIFICIAL ANUS; PERINEAL EXCISION OF RECTUM WITH STUDIES ON SURGICAL ANATOMY, INCLUDING AN ANATOMICAL APPROACH TO THE BASE OF THE BLADDER.

By William C. Lusk, M. D.,

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The following case occurred in the service of Dr. Joseph D. Bryant:

The patient, admitted August, 1903, was a male, Italian, age 55, laborer, suffering from an ulceration of the rectum and anal canal and a small tumor at the anal margin, having had symptoms of diarrhoea with bloody and mucous discharges for four months previous to admission.

Examination showed a hard tumor, the size of an English walnut, superficially ulcerated, at left anal margin; also an annular ulceration about two inches in height, occupying the lower portion of the rectum, and connecting these two seats of disease was a limited area of ulceration in the anal canal. Right inguinal glands slightly enlarged, but otherwise no glandular enlargements anywhere. The pathological report on a specimen taken from the tumor at the anal margin pronounced the growth to be epithelioma. However, another microscopic examination subsequent to operation failed to confirm the diagnosis of malignancy. This diagnostic error emphasizes a recognized fact that simple epithelial growths around the anus are liable to be mistaken histologically for malignant ones.

Accepting the diagnosis of a malignant growth, it was determined to make a permanent inguinal anus and then excise the rectum by the perineal route. In the making of the artificial anus, Dr. Bryant advised dividing the sigmoid loop as low as possible, so as to leave the proximal end of the bowel slack, in order to form a pouch for fecal accumulation. Also he advised closing the distal end at the same time and returning it to the abdominal cavity, and later after excising the rectum to invert and close as well the lower opening of this distal segment. Low section of the sigmoid leaving a loop as a fecal container has been previously practiced by Mayo. The operation in the case here presented was devised so as to make the site of entrance to the abdominal cavity opposite the junction of the outer and middle thirds of Poupart's ligament, corresponding to the locality where the gut passes over the brim of the pelvis, at which situation the bowel could be divided very low, and the proximal end could be brought into place outside with least restraint by the mesosigmoid, as well as with the least disturbance of the normal position of the segment remaining in the iliac fossa. This low exit for the bowel was planned as well with a view of turning the proximal end upwards for a short distance in the tissues of the abdominal wall, so that gravity could aid the intended function of the sigmoid loop as a fecal retainer and at the same time tend to prevent prolapse of the bowel. The extremity of the proximal segment in its upward course was planned to be laid beneath the skin and fascia upon the aponeurosis of the external oblique, as first practiced by Bailey, in order to get the gut in front of a firm structure against which the requisite amount of pressure could be made for control of the anus. Because of the exit of the gut at the site selected, this embedded extremity of bowel could be placed along Poupart's ligament and the anterior superior spine of the ilium, in the neighborhood of which supporting structures the pressure of a truss against the aponeurosis would be most effective and best borne.

OPERATION FOR MAKING ARTIFICIAL ANUS.

An incision 2 inches in length was made parallel with Poupart's ligament, and about 1½ inches internal to it, its upper limit being situated at a point opposite the junction of the outer and middle thirds of the ligament, which is about 1 inch vertically below the level of the line drawn between the anterior superior spines of the ilium. The aponeurosis of the external oblique was incised in the same line. The muscular fibres of the internal oblique and transversalis were separated at the upper angle of the incision, and the transversalis fascia and peritoneum were incised in the line of muscular separation. All the slack in the sigmoid loop, here about 5 inches, was drawn out through the wound and the upper and lower ends of the loop were differentiated. A transverse slit a little over an inch in length was now made through the skin and superficial fascia just above the level of the anterior superior spine and a little within a vertical line passing through the same point, and the bridge of tissue between this and the lower wound was raised from the aponeurosis. The extruded intestine was now clamped about an inch above the site where the descending loop re-entered the peritoneal cavity, by two forceps placed a slight interval apart, the blades of the higher forceps before being applied to the bowel, having been first made to pass through the upper wound and under the bridge of skin and fascia into the lower wound. The bowel was then severed between the clamps with a Paquelin cautery and the ends seared for disinfection. This low section of the bowel left all available slack in the proximal loop to serve as the fecal receptable. The mesosigmoid was split for an inch and a half opposite the seat of section of the bowel so that the divided ends could be separated. The distal end was closed with a purse-string suture supported by Lembert sutures, and returned to the peritoneal cavity. The proximal extremity with its sources of infection controlled as already indicated, was now drawn by means of the clamp attached to it, beneath the cutaneous bridge into the upper wound, to accomplish which it was necessary first to relieve the restraint of the mesosigmoid, by dividing the latter for about ¾ of an inch along its attachment to the free end of the bowel. The length of the segment of bowel extending from the opening in the abdominal wall to the upper wound, was about 2½ inches. The lower flap of peritoneum was sewed to the opposite serous surface of the outgoing bowel. Below the site of exit of the intestine, the edges of the aponeurosis of the external oblique were sewed together so as to form an aponeurotic ring around the bowel at this situation for the purpose of limiting the size of the opening and giving good counter pressure for a truss. The skin was sutured with silk-worm gut. The proximal extremity with clamp left on was sutured with silk to the skin margin of the upper wound. The lower wound was covered with rubber tissue, its edges cemented to the skin with chloroform.

Subsequent course.—Thirty hours after operation patient vomited, and had abdominal pain and distension over colon. The symptoms of obstruction were immediately relieved by loosening the clamp which closed the anus, then inserting a tube into one angle of the bowel, and reapplying the clamp up to the seat of entrance of the tube.

Further course uneventful. After an interval of 6 days, the rectum and right inguinal glands were excised.

EXCISION OF THE RECTUM.

Quénu's* operation by the perineal route was employed.

The coccyx was excised preliminary to the division of the posterior and lateral levator ani fibres, as suggested by Quénu. By this means a longitudinal incision made in the aponeurotic floor of the space from which the coccyx was removed, enabled the finger to be easily passed into a compartment between the levator ani and the rectovesical fascia posteriorly (Fig. 11 and Fig. 13, *Q*), which facilitated the division of the fibres of the levator ani. It was possible to bring down six inches of the bowel below the tip of the sacrum practically without exposing the peritoneal cavity, the peritoneum being stripped up from the anterior wall of the bowel as it was drawn down. A small tear in the peritoneum an inch long did occur during this procedure, which was promptly sutured. The first five inches of the bowel were drawn down after severing the anterior and lateral attachments and stripping the fibrous sheath of the rectum posteriorly only from the lower portion of the sacrum. The additional inch of descent was gained by dividing the rectal sheath across posteriorly, well above the seat of disease, and then severing a posterior median raphé, which is normally found connecting the longitudinal muscular coat of the rectum to the fibrous sheath covering it below the level of the third sacral vertebra (Fig. 14, Sep. 1), and drawing the rectum down inside of its sheath. Five inches of the bowel were amputated, the end was inverted and sewed up, and the stump sutured at the middle of the skin incision which was closed over it. Skin flaps only were brought together. Cigarette drains were employed.

Result.—The patient made an uneventful recovery with complete healing of the wound at first, but soon after developed a sinus,

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*Quénu and Hartman, Chirurgie du Rectum, Paris, 1899.

which on being laid open later (September, 1904), was found to be associated with two retained silk-worm gut sutures. There was no communication whatever with the closed off segment of bowel. Thus this closed undrained segment of bowel left in the pelvis, was found shut off and innocuous after a period of thirteen months. The wound of this last operation, the greater extent of which was freshened and sutured, healed promptly and completely. By thoroughly emptying the bowel with an enema every two or three days, and by the use of a truss made of a water pad held in place by an elastic belt around the crests of the ilium and strap in the perineum, the patient was known to have had absolute control of the bowel contents from the first of April to the middle of June, during which time he did not soil his clothing once. During the following summer, he stated that two or three times he ate something which gave him a copious diarrhoeal discharge and then there was a very slight leakage before he could get to the closet. A very little mucous collects on the fold of gauze placed between the opening and the truss. There is no prolapse of the mucous membrane thirteen months after operation. The patient can pass wind by voluntary retraction of the abdominal wall.

SUMMARY OF CASE.

Making artificial anus.—Intermuscular opening made opposite junction of outer and middle thirds of Poupart's ligament. Sigmoid sectioned low to leave a loop as a fecal container. Distal end closed and returned to peritoneal cavity. Proximal end turned upward on aponeurosis of external oblique in the direction of Poupart's ligament to emerge through the skin a little above the anterior superior iliac spine. Primary union.

Excision of rectum.—Quénu's operation by the perineal route. Five inches of bowel removed. Peritoneal cavity not entered excepting for a small tear. The end of the bowel was inverted and sutured, and the skin flaps closed over it, thus leaving a completely shut-off undrained segment of bowel in the pelvis.

Result.—Artificial anus has been perfectly controllable as regards gas and feces for six months with suitable truss, excepting a very slight leakage two or three times with a copious diarrhoeal discharge. No prolapse of the mucous membrane after thirteen months. The closed undrained segment of bowel was found shut off and innocuous after thirteen months. A perineal sinus, following primary healing, was closed after a number of months by removing from it two retained silk-worm gut sutures.

SURGICAL ANATOMY.

The following original illustrations of studies in surgical anatomy relating to perineal excision of the rectum are presented. Figures 4 to 10 are photographs with lines emphasized by the artist, illustrating an anatomical plane of separation between the rectum and the adjacent structures of the urethral triangle. The illustrations showing this plane of separation were completed by the writer prior to his knowledge of the publication on this subject by Gosset and Proust,* to whom credit is due for having established the operation of approach to the base of the male bladder on a definite anatomical basis. The writer cannot find that any investigator previous to himself has described the attachment sometimes present of the longitudinal coat of the rectum around the apex of the prostate.

Figure 3.—*A* is a small anterior bundle of external sphincter fibres which laterally adjoins the mediam attached anterior sphincter fibres and in the middle line passes beneath them, forming with the corresponding muscle of the opposite side a loop which can be designated as the *superficial,* in contradistinction to the deeper anterior sphincteric loop *BC,* Figure 6. In this illustration the median attached anterior fibres of the external sphincter have been separated from *A* showing the aponeurotic structure *D* beneath (see Fig. 14, *D*). *D* extends laterally around the anal canal, separating the external from the internal sphincter. On its inner surface it receives the insertion of the longitudinal muscular coat of the rectum, and above, it is continuous all around with the recto-vesical fascia. In front the recto-vesical fascia is an important guide in approaching the base of the male bladder. As it is followed upward in this situation, it is found to split into two layers, an anterior passing to the lower portion of the central tendon of the perineum (Fig. 14 *CT*) and its laterally attached transverse perineal muscles (Fig. 8, *G*), and a deeper layer (Figs. 9, 12 and 14, *H*), which covers the recto-urethralis muscle and is attached around the base of the triangular ligament in a U-shaped line, corresponding to the origin of this muscle (see under Fig. 9). The muscular bundle *B* after receiving a fibrous attachment from the ramus passes in a fascial compartment to the front of the anal canal where it forms with the corresponding muscle of the opposite side a considerable portion of the sling *BC*, Fig. 6. This bundle, encircling the anal canal, can be regarded as sphincteric. The bundle *C* has been dissected away from *B* with which its fibres normally lie in close association. It corresponds to the muscle described by Harris* as the puborectalis. It is a connecting link between external sphincter and levator ani. The arrangement of its anterior fibres with relation to these muscles varies in different dissections, the fibres sometimes following more the arrangement of the bundle *B* when the joint action of the muscles of the two sides would be chiefly sphincteric in character, and again associating most of its fibres with the levator ani, so that any picture of its anterior terminal distribution would represent but one appearance of it. However, it seems constantly to send some fibres to the deep sphincter sling *BC*, Figure 6, and generally for the rest of its course either to blend closely with, or to follow the course of (Fig. 13, *C*), the anterior bundles of the levator ani (Figs. 12 and 13, *M*). If the latter condition present, a plane of cleavage anteriorly can be demonstrated between it and the levator ani proper, but if the fibres blend, this bundle can be isolated only by a false dissection. In rectal excision the bundles *B* and *C* can be raised out of their bed antero-laterally (Fig. 11) by a dissection beginning laterally beneath one side of the sling *BC*, Figure 6, first stripping the sling outwards a half inch and splitting the upper margin of its fibrous compartment, then passing a scalpel handle directly outward and a little upward through the split in the fascia, across the base of the anterior bundle of the levator ani, thus getting beneath the muscular bundle *C*, and, when the tissues are separated, turning the instrument backward and outward in the direction of the ramus and forcing it through the anal fascia into the ischio-rectal fossa. *B* and *C* can then be severed in this line of separation on either side, and the anterior bundles of the levator ani (Fig. 12, *M*), will come into view. The resulting liberation of tissues facilitates access to the base of the bladder in rectal excision.

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*Gosset et Proust. Le Muscle Recto-urethral. Son importance dans les operations par voie périnéale, en particulier dans la prostatectomie. Bulletin de la Soc. Anat. de Paris 77, 1902, p. 425.

Proust. La Prostatectomie. Paris, 1904.

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*Harris, M. L. The repair of old lacerations of the pelvic floor. Jour. Am. Med. Assn. Dec., 1899., p. 1450.

Approach to base of bladder (Figs. 4 to 10).—Make an inverted *Y* incision, the arms of the *Y* diverging half an inch in front of the anal margin, and dissect out the anterior fibres of the external sphincter.

Figure 4 shows the superficial anterior loop of the external sphincter (*A*) separated from the medain-attached anterior sphincter fibres (*ES*). The latter are hooked up from *A* and the aponeurosis (*D*) beneath. If the sphincter is to be saved it should be cut as close as possible to its anterior attachment.

Figure 5 shows the median-attached anterior fibres of the external sphincter (*ES*) cut across and the loop *AA* (here consisting of two separate bundles) dissected forward on the fascia until, through the fascia thus laid bare, there is seen a transverse muscular band, which is the deep anterior loop of the external sphincter (*BC*, Fig. 6). The black line indicates the site of an incision in the delicate fascia covering this muscle, for entering its compartment (Fig. 14, *BC*). In one dissection a distinct compartment for this muscle was lacking, the muscle lying directly beneath the anterior fibres of the external sphincter, with practically no fascia intervening. In another dissection this muscular band was entirely absent, the transverse incision through the fascia beneath the loop *A* then opening directly into the double compartment (Fig. 7).

Figure 6 shows the fascial compartment lodging what can be termed the *deep* anterior loop of the external sphincter (*BC*) opened up, and the contained muscle stripped upward, exposing a fascial bed beneath.

The muscular band is about half an inch broad and is made up of the bundle *B* and some fibres from *C* (see under Fig. 3). At its upper border it fuses with the lower fibres of the transversus perinei muscles, for the location of which it can serve as a guide. It can be loosened up on either side by stripping it from the floor of the fascial compartment in which it lies, with the handle of a scalpel, thus gaining additional room for the subsequent steps of access to the base of the bladder. When the muscle has been completely reflected, at its upper margin the apex of the central tendon of the perineum is reached, which sometimes now comes into view turned upward with the muscle. The central tendon of the perineum (Figs. 7 and 12 *K*, and Fig. 14, *CT*) is a triangular-shaped extension from the middle of the base of the superficial layer of the triangular ligament, pointed downward, from about ⅜ to ⅝ of an inch in length, which receives laterally transverse perineal muscle fibres and supports the bulb. The next step is the making of a transverse incision as indicated in the figure, merely through the fascia in the floor of this compartment, at its upper limit just below the attached border of the reflected tissue. Tension on the two planes will emphasize the angle where they meet.

Figure 7 shows the fascia divided and the next compartment (Fig. 14, *DC*), which is a double one, opened up. The compartment extends forward to the base of the superficial layer of the triangular ligament, and by opening it up the bulb is lifted from the rectal wall. Entrance to it is gained by pushing backward with a scalpel handle on either side of the median line the muscle fibres which present when the fascia is cut, hugging with the instrument the roof of the space, which is the central tendon of the perineum (*K*) and its laterally attached transverse perineal muscles. At the beginning of the separation of these planes a little resistance may be encountered, which occurs when the incision in the fascia has been made a little low. There is an easy plane of cleavage between these muscle fibres and its central tendon of the perineum, particularly near the median line. The picture of the compartment thus laid open is one presenting two spaces separated by a median septum, from which muscle fibres radiate downward and laterally. The lateral of these fibres are associated with those of the anterior bundles of the levator ani (Fig. 12, *X*), of which they are practically a part. The depth of the space is usually from about ⅜ to ⅝ of an inch, and the median septum distinctly marked as in the picture. In one dissection, however, the depth was found to be only about 1-3 of an inch and the septum an insignificant structure, which tore readily, these differences causing temporary perplexity in the dissection. The anatomy, however, did not otherwise differ from the normal and the separation of the upper planes was made as usual, though complicated by a rather small opening of access through the recto-urethralis muscle (Figs. 10 and 14, H^1), owing to its correspondingly short vertical dimension. Too much force must not be used in cleaving the planes of this compartment, or the instrument may pass beyond the vault of the compartment, which is a fibrous fold (Fig. 9), and gives a moderate resistance.

Figure 8 presents the same compartment as Figure 7 and shows its relations to the urethra and bulb. Structures of the anterior urethral compartment on the left side of the dissection have been removed, to show the triangular ligament, and the roof *G* of the double-barreled compartment continuous with the lower edge of its superficial layer. The roof of the double compartment is formed of the central tendon of the perineum with its laterally attached transverse perineal muscles covered with fascia. The compartment of the triangular ligament is shown opened up and the membranous urethra (+) covered by the compressor urethræ muscle, has been projected into the opening on the tip of a sound introduced within its lumen, which has been turned laterally in situ, thereby deviating the membranous urethra from its normal medain position. The situation of the bulbous portion of the urethra in which the sound lies can be traced in shadow (*Y*). The artery of the bulb (*AB*) is seen within the compartment of the triangular ligament along the base of its superficial layer. The next step is to cut the septum of the double compartment with scissors, near its rectal attachment, and then, with a scalpel handle, push the cut muscle fibres away from the median line toward either side (Fig. 12, *X*), thereby exposing in the floor of the space a fascia (Figs. 9, 12 and 14, *H*), which is the anterior portion of the recto-vesical fascia (see under Fig. 3). Above, this fascia forms a reflection at the base of the central tendon of the perineum, so that, when this compartment is opened up, it presents a fibrous vault.

Figure 9 shows the fibrous vault, the roof of which is the central tendon of the perineum (*K*) with its laterally attached transverse perineal muscles covered with fascia, and the floor of which is the recto-vescial fascia (*H*). The dome of the vault is at the base of the superficial layer of the triangular ligament (*cf*, Fig. 14, *DC*). The roof of the compartment has here been split longitudinally on the left side up to the artery of the bulb (*AB*), which indicates the situation of the base of the superficial layer of the triangular ligament.

The recto-vesical fascia in this situation covers in the recto-urethralis muscle (Figs. 10 and 14, H^1). The upper attachment of this muscle is along two converging lines which, starting from the sides of the prostate a little above its apex, cross the basal layer of the deep urethral compartment (Figs. 10, 12 and 14, *I*), and meet at the middle of the base of the superficial layer of the triangular ligament, outlining a U-shaped area (Figs. 10 and 12, *I*) in front of the prostate, with which the rectum is in apposition, and which corresponds to the situation of the membranous uretha (Fig. 10, dotted line) and a very little of the bulb, lying superior to it. The length of the U-shaped area varies from less than a half to three-quarters of an inch. The recto-urethralis muscle terminates below either by fusion with the longitudinal coat of the rectum, or if the longitudinal coat be absent in this situation as sometimes occurs, it terminates in the aponeurotic structure between the sphincters (Fig. 14, *D*). The length of the recto-urethralis muscle before fusing with the rectal structures has seemed to about correspond to that of the median septum of the double compartment (Fig. 7), so that when this septum is longer the muscle is longer and *vice versa*. The deep surface of the recto-urethralis muscle above its junction with the bowel has a fibrous texture, and from this aspect of it, the rectal wall can be peeled away with more or less ease. The plane of cleavage beginning at this situation almost always is continued directly upward between the rectal wall and the basal layer of the triangular ligament or U-shaped area (Figs. 10 and 14), and in a considerable number of instances it extends on unobstructedly into the prerectal space.

The black lines in the illustration indicate the route of access to the deeper plane of separation. The transverse line indicates the site of an incision through the recto-vesical fascia only. This should be made close to the central tendon of the perineum (*K*) corresponding to the upper portion of the recto-urethralis muscle beneath, at which situation this muscle can be penetrated with greatest directness of approach to reach the next objective point which is the plane of cleavage between the rectum and the basal layer of the triangular ligament or U-shaped space. The upper limit of the median septum of the double compartment is a good guide to the upper level of attachment of the recto-urethralis muscle in the median line, which point can be located as the septum is cut. The vertical black line indicates the site of entrance through the recto-urethralis muscle, which entrance should be effected by splitting the fibres of the muscle with delicate blunt force, as by the use of the corner of a square-ended scalpel handle.

The direction of the force employed should not at first be downward and backward in the direction of the fibres of the muscle, but rather upward and backward in the direction of the basal layer of the triangular ligament (Fig. 14), which should be closely hugged by the separating instrument, the latter thus pushing the rectal wall away *primarily* from the base of the triangular ligament, *after* which it should continue the separation of the rectum from the deep surface of the recto-urethralis muscle. The opening can then be enlarged by cutting the recto-urethralis muscle transversely. A lower point of primary entrance through the recto-urethralis muscle than the one here mentioned is likely to endanger the integrity of the rectal wall, particularly if the longitudinal coat is lacking here. Gosset and Proust speak of the importance of cutting the recto-urethralis muscle close to the bulb, as well as the change in direction of the bowel wall at the site of its meeting the recto-urethralis muscle, forming an elbow, which anatomical fact should be kept in mind in penetrating this muscle and separating back the rectal wall beyond.

Figure 10.— The recto-vesical fascia (*AF* or *H*, Fig. 9) has been cut transversely clear across and the opening stretched by traction to show the recto-urethralis muscle (H^1) beneath, which latter has been split longitudinally in accordance with the incision indicated in Figure 9, and the rectal wall (*V*) beyond has been separated back by blunt pressure from the U-shaper area (*I*) and base of the bladder. The anatomical arrangement of the separated structures beneath H^1 is seen up to the transverse oval opening which is the entrance at the apex of the prostate to the fascia-lined infravesical prerectal space (*U*). The relation of the rectal coats to the deep surface of the recto-urethralis muscle and the U-shaped area has been found to vary. In this figure a fascial layer covering the longitudinal coat (*V*) of the rectum was encountered immediately beneath these structures, was readily peeled back from them, and the separation was continued upward unobstructedly until the prerectal space (*U*) was entered. In Figure 12 circular fibres lay immediately beneath the recto-urethralis muscle, the longitudinal coat had a few fibres attached over the U-shaped area, and a firm adhesion to the apex of the prostate, which had to be cut. Again, longitudinal fibres may be met beneath the recto-urethralis muscle, which readily strip from the U-shaped area up to the apex of the prostate at which situation an adhesion is found (Fig. 14, *Z*). In one dissection the circular coat alone existed all the way up to the apex of the prostate, which was readily peeled from the U-shaped area; opposite the apex of the prostate the fibres of the longitudinal coat were found to terminate, but they had no prostatic attachment, so that simple blunt dissection here again gained direct entrance into the infravesical space. In another case the longitudinal coat had an attachment to the very front of the U-shaped space, above which there was an easy plane of separation; below the attachment of the longitudinal coat, beneath the recto-urethralis muscle, a few fibres of the circular coat were met which readily spread showing the submucous coat beneath. In still another instance the recto-urethralis muscle was absent, the incision in the recto-vesical fascia then opening directly down onto the longitudinal coat of the rectum, which was readily peeled back from the anteriorly situated structures all the way up to the prerectal space. The surgical importance of the foregoing resolves itself into the recognition of the attachment of the longitudinal coat to the apex of the prostate or possibly to the front of the U-shaped space, when present, a failure to do which might easily result in a tear into the rectum caused by the blind employment of blunt force directed against this obstacle or between the rectal coats. The attachment of the longitudinal coat to the prostate when present is around its apex and laterally on either side for about half an inch. Only the central portion attached to the apex is divided in gaining access to the base of the bladder, the lateral prolongations being cut, together with the other antero-lateral attachments of the bowel, as a later step in rectal excision (see under Fig. 13). The attachment should be cut close to the prostatic tissue which can be recognized by its firm resistance.

The ease with which a prostatic attachment of the longitudinal coat can be located varies with the anatomical condition found. If the condition is that in which a thin layer of the longitudinal fibres of the rectal wall is continued downward from the prostatic attachment behind the U-shaped space and the recto-urethralis muscle (Fig. 14), the stripping back of the bowel can then be effected as far as to the adhesion which is known to have been met when the layers cease to separate. If, however, the circular coat of the bowel lies immediately beneath the U-shaped space and the recto-urethralis muscle, the location of the prostatic attachment of the longitudinal coat may be perplexing, since, when the apex of the prostate is reached, the plane of blunt separation then passes between the circular and longitudinal coats entirely posterior to the adhesion, and consequently the separating instrument meets no line of distinct opposition. A too high separation between these layers of the rectum is likely to bring the instrument down onto the submucous coat of the bowel. When, therefore, the separation of the tissues has been carried to above the prostatic apex and the prerectal space found not to have been entered, an attachment of the longitudinal coat to the apex of the prostate should be suspected. In this event the tissues passing upward from the apex of the prostate should be pulled downward into a fold which is snipped through with a scissors, when, if this fold be the longitudinal coat, entrance through it into the prerectal space can thereupon be gained by blunt force at the seat of cutting. The prerectal or infravesical space is recognized by its having a complete fascial lining (Figs. 13 and 14, *U*), between the layers of which is a delicate areolar tissue which readily gives way before the entering finger. The space assumes the pyramidial shape as described by Quénu when it is completely opened up. The white dotted line in this figure indicates the situation of the membranous urethra which lies a safe distance (⅛ to ¼ inch) above the basal layer of the triangular ligament (*I*), fibres of the compressor urethræ muscle intervening.

Figure 11.—The grooved director has been inserted in the picture to indicate how the bundles *B* and *C* (Fig. 3) can be raised antero-laterally from the anterior bundle of the levator ani (Fig. 12, *M*), beneath and cut across in rectal excision as described under the consideration of Figure 3. The finger passed through a lateral incision in the floor of the space from which the coccyx has been removed, enters a postero-lateral fascial compartment (Fig. 13, *Q*), and meets a fold which limits the compartment anteriorly. The incision of entrance into the compartment is made a little to one side of the median line since a median septum (Fig. 14, Sep. 2), divides the compartments of the two sides. The interrupted line shows the site of section of the posterior and lateral levator ani fibres in rectal excision. When this muscle has been cut along the finger to the anterior limit of the compartment, the fibres in front can be raised directly off the fascia and divided.

Figure 12.—The bundles *B* and *C* (Figs. 3 and 11) have been divided antero-laterally, exposing the anterior bundles of the levatores ani (*M*) beneath. In this dissection distinct planes of cleavage between the bundles *C* and the anterior bundles of the levatores ani were found. The double compartment (Fig. 7) between the central tendon of the perineum (*K*) and the recto-vesical fascia (*H*) has been opened up. The cut muscular edge (*X*) framing *H* laterally and below, is that formed by the reflected fibres after severance of the median septum of the double compartment (see under Figs. 7 and 8). The median split in the recto-vesical fascia and the recto-urethralis muscle beneath it, shows the circular coat (*L*) of the rectum separated back a little ways from the under surface of the recto-urethralis muscle and from the basal layer of the deep urethral compartment (*I*), which latter here receives the insertion of a few fibres of the longitudinal coat. The attachment of the longitudinal coat to the apex of the prostate is cut showing the entrance to the infravesical or prerectal space, just behind the letter *I*. The anterior fibres of the levator ani (*M*) here arise on either side from the posterior surface of the body of the pubis and along the descending ramus; they pass above the triangular ligament, and are inserted into the recto-vesical fascia at the side of the membranous urethra and adjacent portion of the rectum, and below fuse with the longitudinal muscular fibres of the rectum. Anteriorly these fibres are continuous with those (*X*) in the floor of the double compartment (Fig. 7), which converge toward the median septum.

Figure 13 shows the principal antero-lateral attachments of the rectum which are severed in rectal excision. They are on either side, the anterior bundle of the levator ani (*M*), and the recto-vesical fascia (*P*) reflected from the side of the prostate and the vesiculis seminalis. Also in instances where there is an insertion of the longitudinal coat of the rectum around the apex of the prostate the laterally attached of these longitudinal fibres must receive consideration in this connection; since, when this attachment be present, it holds the rectal wall closely to the sides of the prostate not allowing it to be stripped back, so that the line of severance must then be made very close to the prostate or the rectal wall will be cut through. When, on the other hand, there is no prostatic attachment of the longitudinal coat, on opening up the infravesical space with the finger, the rectum recedes sufficiently so that the antero-lateral attachments can be severed without danger of entering the bowel. The recto-vesical fascia has been here opened up in the line of its severance in rectal excision showing the middle hemorrhoidal artery (*S*) beneath, which is found and tied by Quénu in this situation before its division; also there is shown the branch to the prostate (*T*), and more deeply the infravesical or prerectal space (*U*). The letter *U* is placed over the base of the prostate which is entered at this situation by its artery. *W* is placed on the fibrous structure covering the vesiculis seminalis. *Q* is the postero-lateral compartment of the recto-vesical fascia showing where the finger introduced through the tissues beneath the coccyx (Fig. 11) meets the fold limiting the space anteriorly.

Figure 14 shows in diagram anteriorly the fascial compartments which are opened in reaching the base of the bladder. *H'* represents the recto-urethralis muscle which is covered in front by the recto-vesical fascia (*H*). Beneath its fibrous deep surface is shown an interval representing the plane of cleavage between this layer and the rectal wall. The plane of cleavage here extends upward between the rectal wall and the basal layer of the triangular ligament (*I*) to the prostatic attachment (*Z*) of the longitudinal coat. Above the prostatic attachment is the fascia-lined prerectal space (*U*). Posteriorly some of the rectal attachments are shown. *R S* is the retrorectal space forming a fascial compartment between the sacrum and bowel down to the tip of the sacrum where the fascial boundaries of this space from all sides come together in a point. For the lateral boundaries of this space see Figure 15.

Figure 15, *abc*, is the V-shaped line of fascial attachment of the bowel to the anterior surface of the sacrum, the attached folds forming the lateral boundaries of the retrorectal space (Fig. 14, *RS*). This compartment can be entered after excising the coccyx and cutting through the next layer of tissue beneath it down to the recto-vesical fascia, by the employment of blunt upward pressure with the finger at *b*, along the median line in front of the sacrum. The sacral attachments below the level of the fourth sacral foramina can be easily torn through with the finger, by doing which a considerable opening at the lower end of the retro-rectal compartment can be made. The sacral attachments cannot be torn by blunt force higher than this level because here they are tougher and likewise are joined laterally by the layers of recto-vesical fascia (ef and $e^{I}f^{I}$) attached along the upper borders of the lessor sacro-sciatic ligaments, thus forming a rectangualr fold (f and f^{I}) on either side around the fourth sacral foramen, within which lie the branches of the fourth sacral nerve going to the anus. Stripping upward in the retro-rectal space will easily raise the fascial layer behind the bowel from the fronts of the bodies of the sacral veterbrae up to the promontory. The lateral attachments can be distinctly felt on either side from within the space. On the cadaver in rectal excision after the recto-vesical fascia has been divided antero-laterally along the side of the prostate and vesiculis seminalis on either side, and then postero-laterally below its lines of attachment to the lesser sacro-sciatic ligaments as far as to the points f and f^{I}, the finger can then be insinuated externally to the folds fa and $f^{I}c$ readily cleaving them from the adjacent externally situated structures. Then by division of these separated sacral folds, the middle hemorrhoidal vessels having previously been ligated on either side, the bowel can be drawn down to the full extent allowed by the length of the superior hemorrhoidal vessels, a distance of seven to eight inches below the level of tip of the sacrum, without any division at all of the lateral peritoneal folds of the rectum, or any opening at all into the peritoneal cavity, unless one at the lower extremity of the *cul de sac* in case the latter cannot be stripped high enough from off the front of the rectal wall to be above the seat of amputation.

SUMMARY OF SURGICAL ANATOMY.

Approach to base of bladder.—The median attached anterior fibres of the external sphincter are cut close to their insertion (Fig. 4), and the aponeurosis (*D*) exposed beneath (Fig. 5). This aponeurosis is directly continuous above with recto-vesical fascia (Fig. 14) which, where it forms the floor of the compartment containing the deep anterior loop of the external sphincter (*BC*, Fig. 6), is the guide to the double compartment (Fig. 7). The muscle bundles (*B* and *C*, Fig. 3) can be raised antero-laterally (Fig. 11) and cut (Fig. 12). The septum of the double compartment is divided, and the muscle fibres in the floor of the space, thereby freed from their median attachment, are separated laterally exposing the recto-vesical fascia (*H*, Figs. 9, 12 and 14) beneath, which covers the recto-urethralis muscle (H^1, Figs. 10 and 14). This fascia is severed transversely high up (Fig. 9) close to the central tendon of the perineum (*K*). The upper limit of the septum of the double compartment corresponds to the level of the upper limit of the recto-urethralis muscle beneath it, and is therefore a guide to this point. The recto-urethralis muscle is penetrated at its upper portion by blunt force directed *first* upward and backward to separate the rectal wall from the basal layer of the triangular ligament (*I,* Figs. 10, 12, and 14), *after* which the rectum is separated from the deep fibrous surface of the recto-urethralis muscle. The recto-urethralis muscle can be cut transversely to gain more room for access. The infravesical or prerectal space can now be entered either directly by blunt force, or after first cutting an attachment of the longitudinal coat of the rectum to the apex of the prostate, if present (see under Fig. 10).

Other Anatomy.—If the coccyx be excised, the finger can be introduced through a lateral incision in the floor of the space from which it was removed, above the levator ani, and this muscle raised and cut along the finger to its anterior fibres (Fig. 11). The antero-lateral attachments of the rectum are, the anterior bundles of the levator ani (*M,* Figs. 12 and 13), the recto-vesical fascia (*P,* Fig. 13), and, when present, the lateral portions of the attachment of the longitudinal coat of the rectum to the prostate. The middle hemorrhoidal vessels (*S,* Fig. 13) are found and tied high up antero-laterally. The recto-vesical fascia is severed postero-laterally on either side below its attachment along the upper margin of the lesser sacro-sciatic ligament, from the spine of the ischium to below the fourth sacral foramen ($e\ f$, $e^1\ f^1$, Fig. 15). The rectum and its posteriorly attached structures are raised from the anterior surfaces of the bodies of the sacral vertebrae by blunt separation. Finally, on the cadaver, the fascial folds $f\ a$ and f^1c, Fig. 15) can be separated from the externally situated structures and cut, thereby allowing the bowel to be pulled down to the full extent allowed by the length of the superior hemorrhoidal vessels without opening into the peritoneal cavity unless at the tip of the *cul de sac,* which latter can be freed from the anterior rectal wall a short distance by blunt upward stripping.

My thanks are due to Mr. O. G. Mason for his careful photography, and to Mr. K. K. Bosse, the artist, in preparing the illustrations.

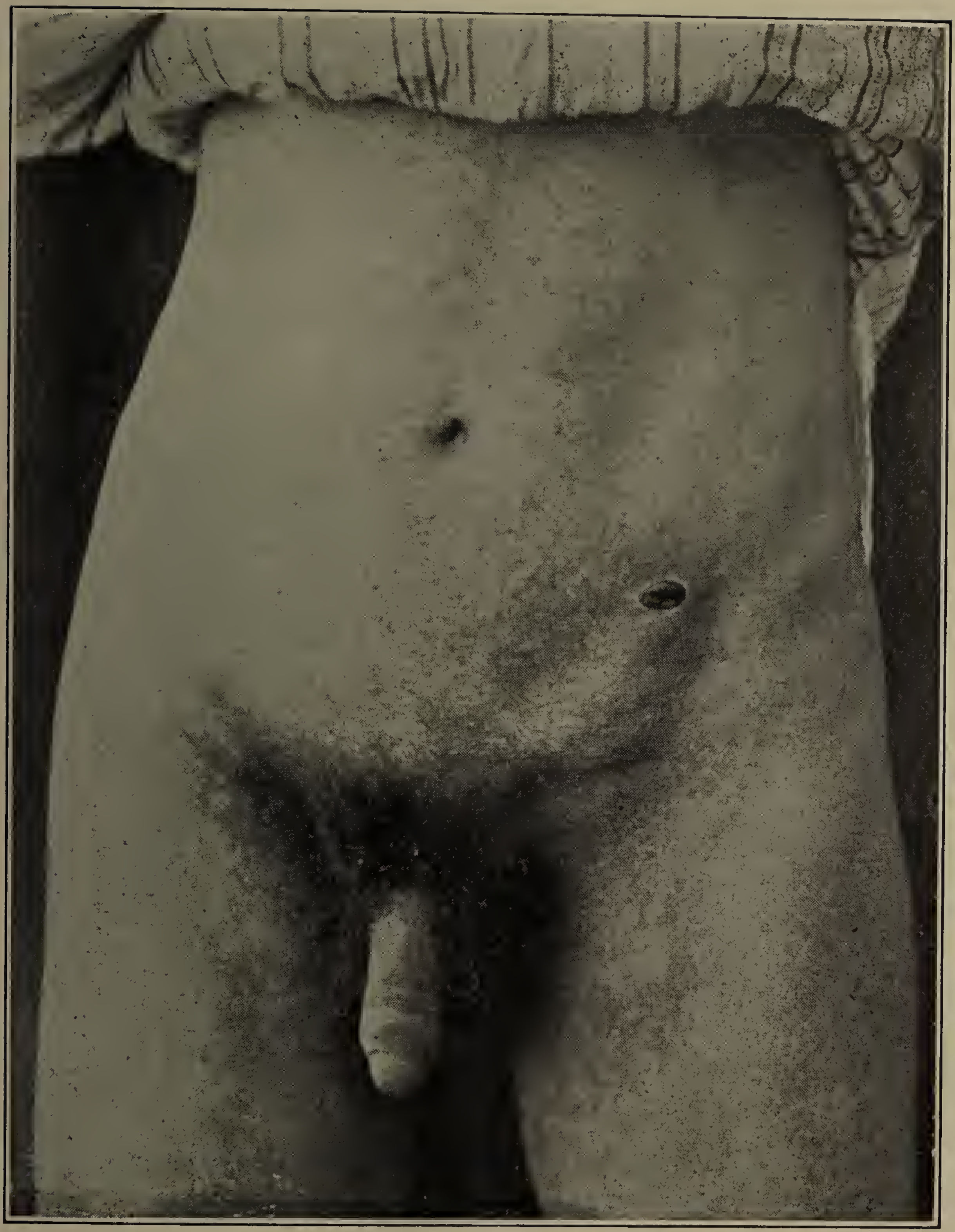

Fig. 1. Permanent artificial anus.

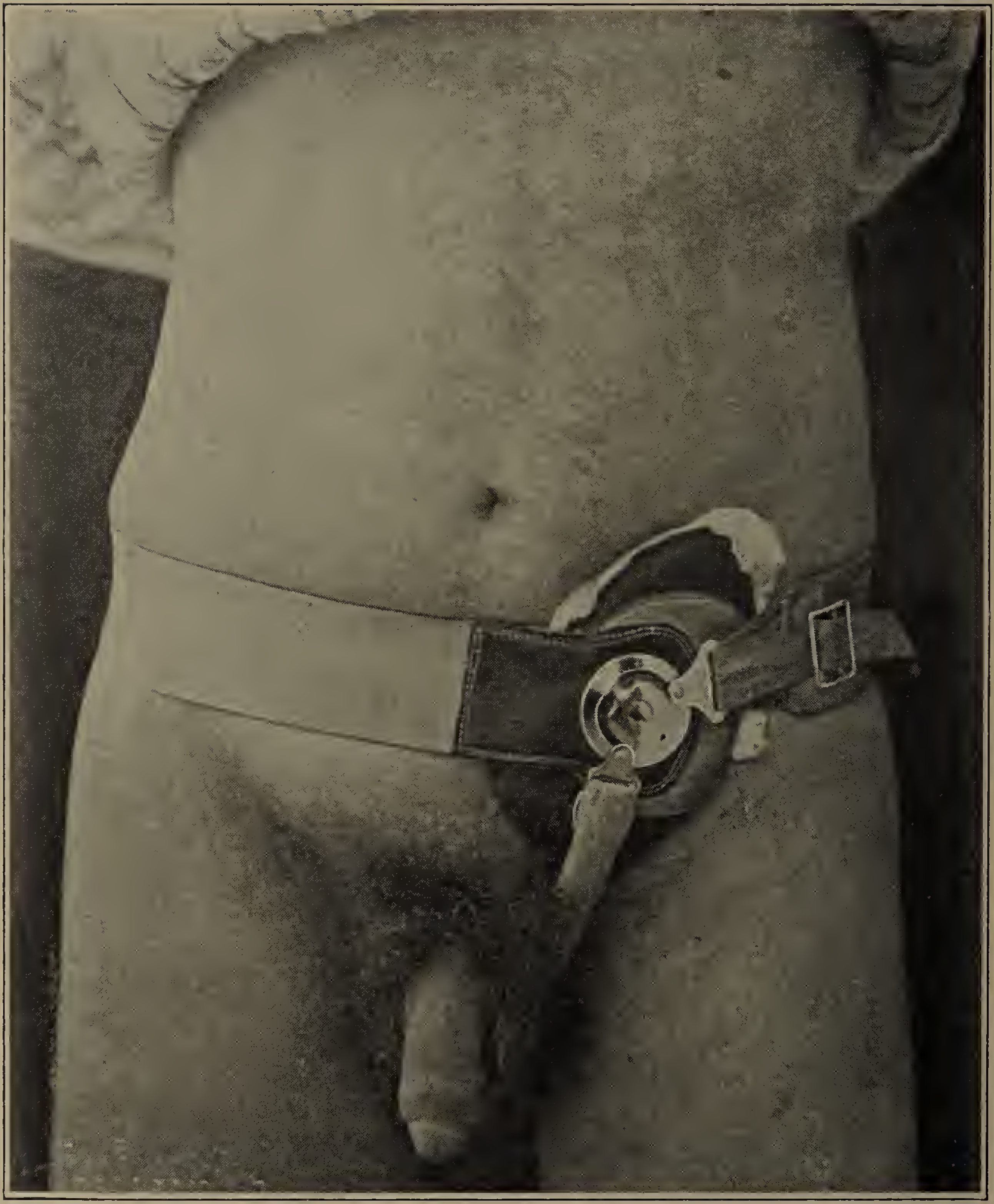

Fig. 2. Truss in place over anus.

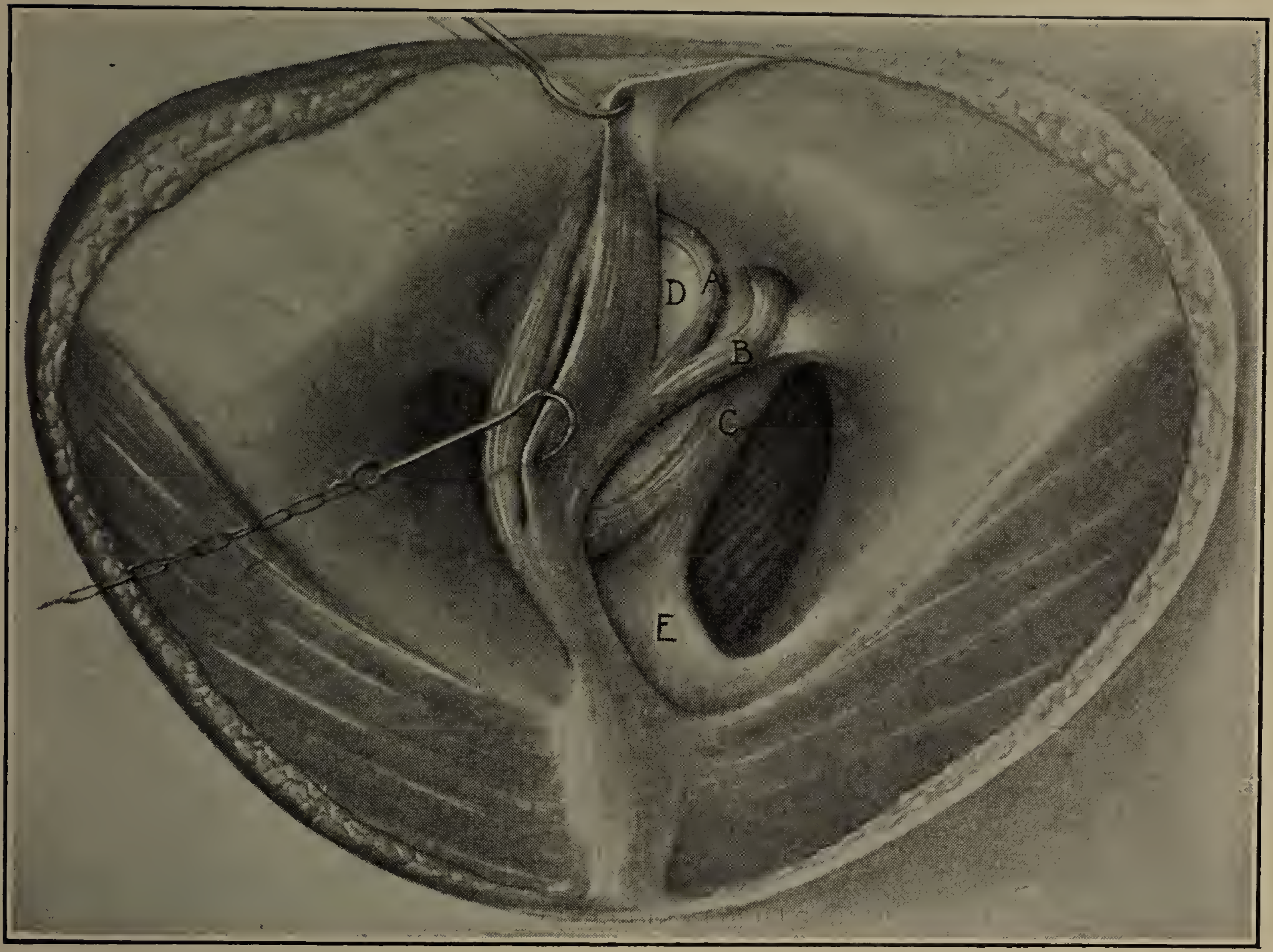

Fig. 3. (drawing from photo.) The left edge of external sphincter is turned down and drawn toward opposite side. A superficial anterior loop of external sphincter fibres going beneath the median-attached anterior fibres from which it has been separated. In the interval is seen D which is the aponeurosis between the external and internal sphincters. B deep anterior sphincteric fibres going to receive a fascial attachment from the ramus whence they turn forward and inward to form part of the bundle BC, fig. 6. C pubo-rectalis muscle (cf. fig. 13), or connecting fibres between external sphincter and levator ani, separated from B. E anal fascia.

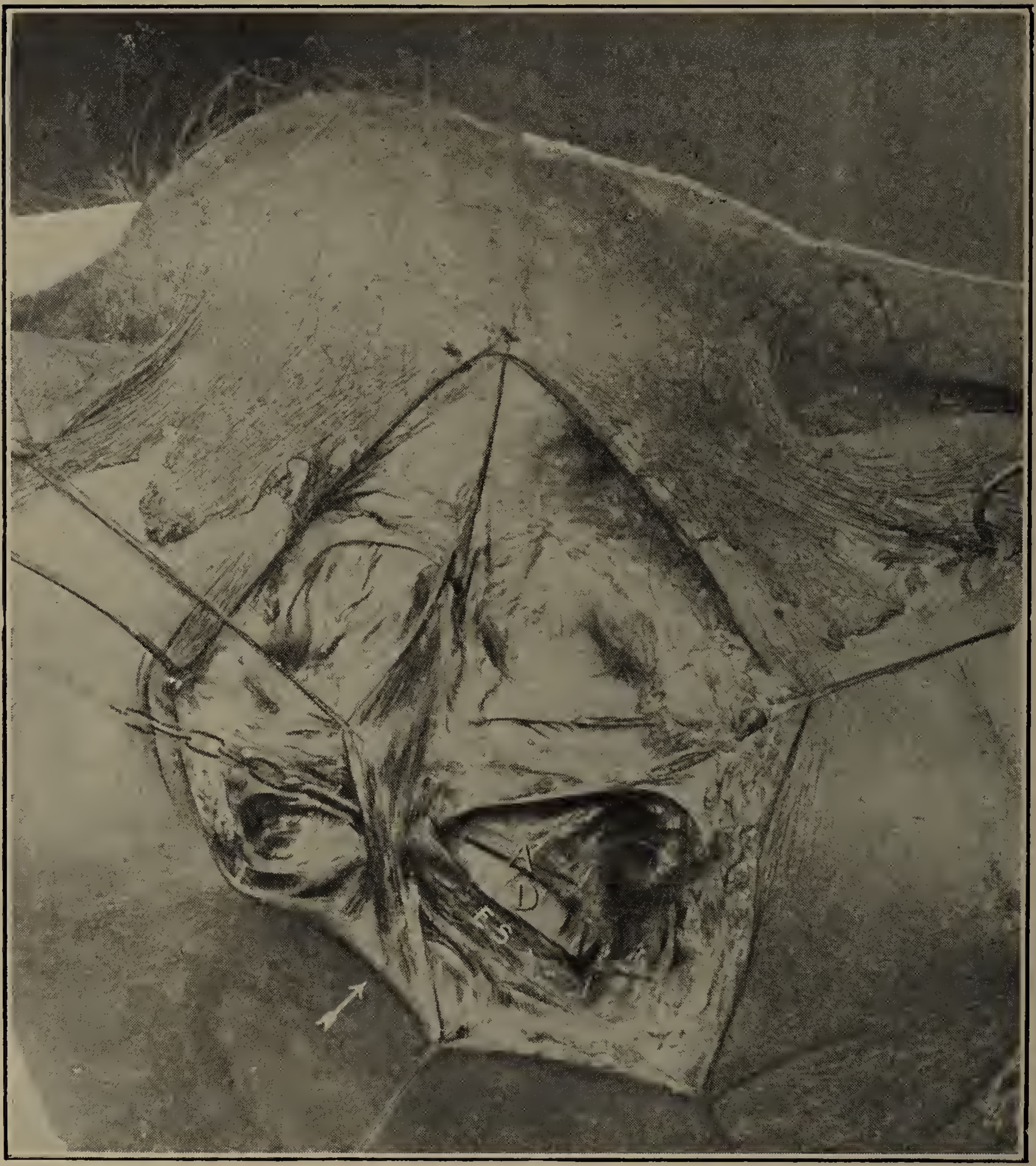

Fig. 4. (photo.) A superficial anterior loop of external sphincter separated from median-attached anterior sphincter fibres (ES). The latter are hooked up from A and from the aponeurosis D. The arrow points toward the anal opening.

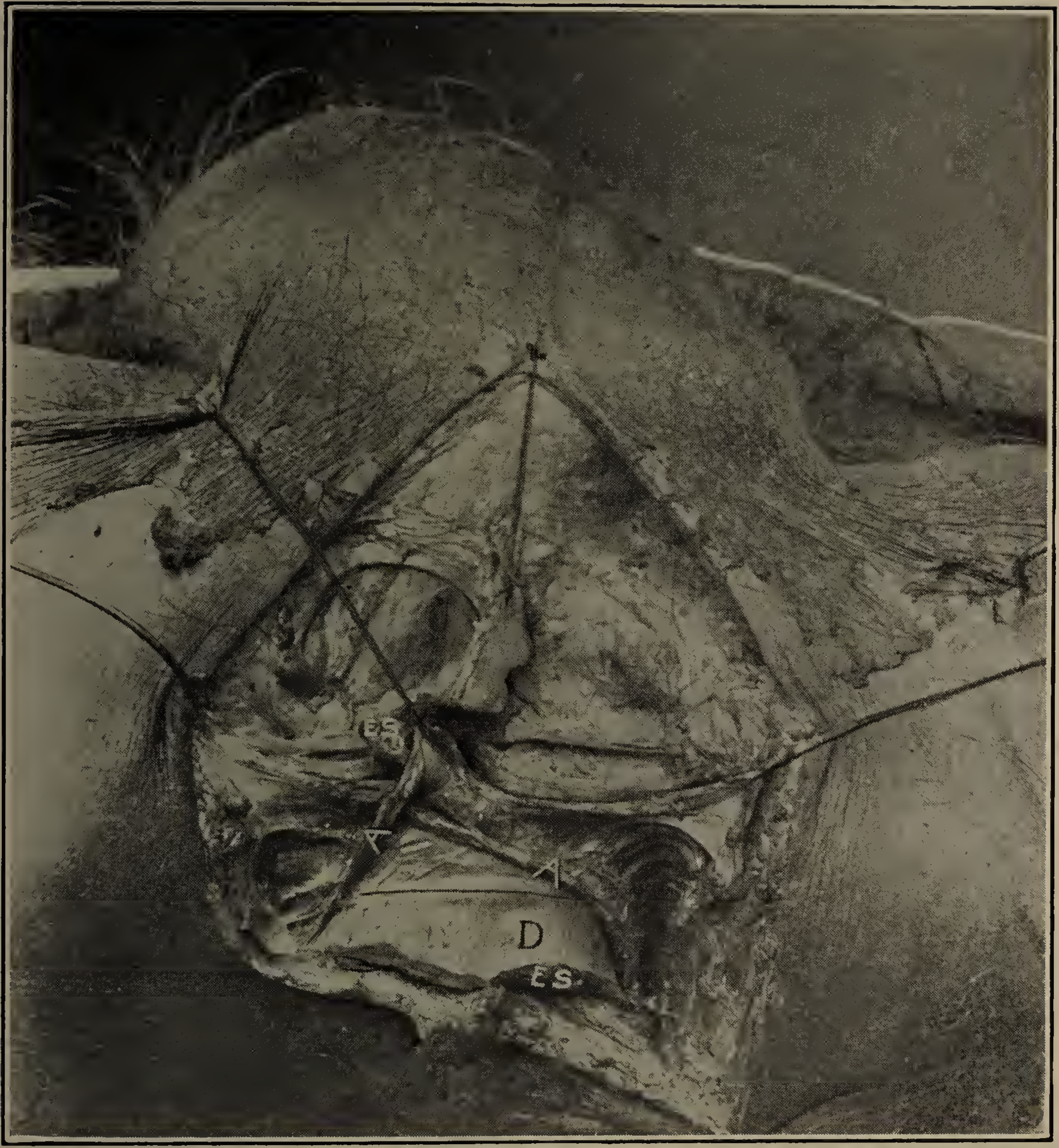

Fig. 5. (Photo). Median-attached anterior fibres of external sphincter (ES, E S) cut and reflected showing beneath, the superficial anterior loop (A A) of the external sphincter which is dissected upward. This loop here consisted of two separate muscle bundles. Black line is incision through fascia over lower border of the deep anterior loop of the external sphincter (BC, fig. 6) seen through it. D aponeurotic layer between external and internal sphincters.

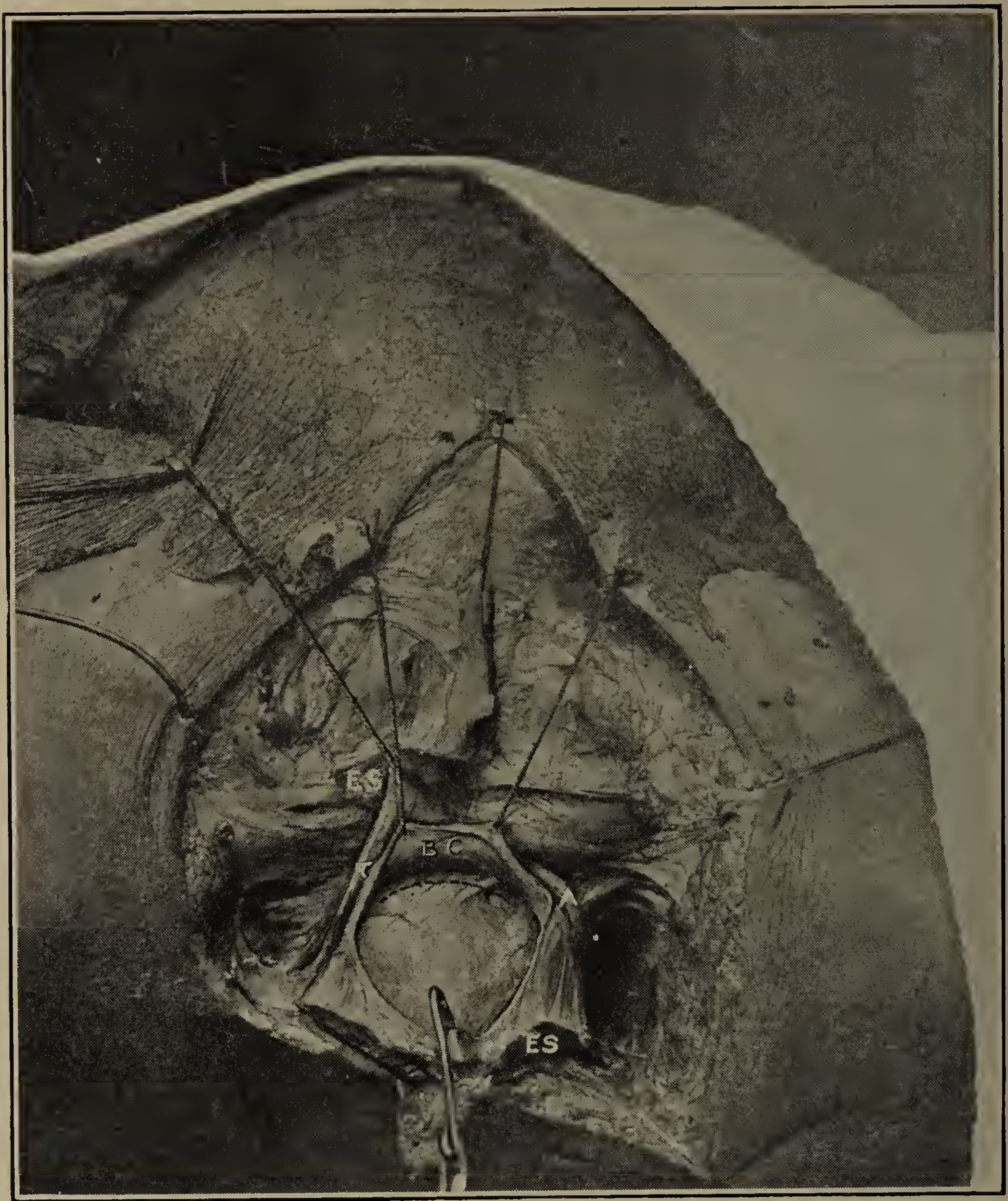

Fig. 6. (Photo). BC deep anterior loop of external sphincter (sometimes absent) turned upward out of its fibrous compartment (fig. 14, BC). The reflected tissue here did not include the extremity of the central tendon of the perineum. Dotted line is the incision through fascia only, in the floor of the compartment just below its angle of junction with the reflected muscle. ES, E S median-attached anterior fibres of the external sphincter cut across.

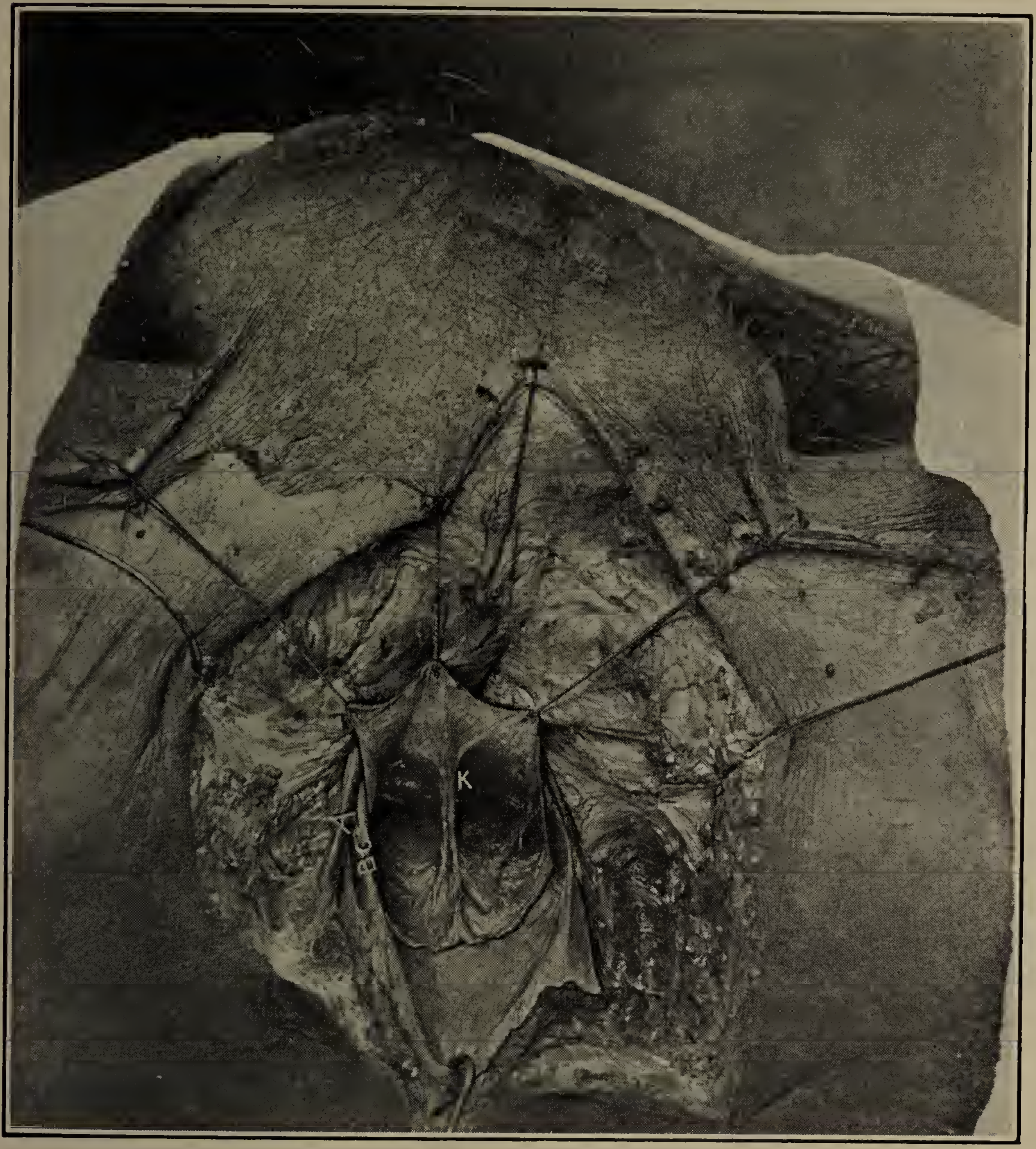

Fig. 7. (photo.) Double compartment (fig. 14, DC) beneath central tendon of perineum (K) opened up. By opening it up the bulb is lifted from the rectal wall. The muscle fibres in the floor radiate downward and laterally from the median septum, laterally being associated with the fibres of the levatores ani (cf. fig. 12, X).

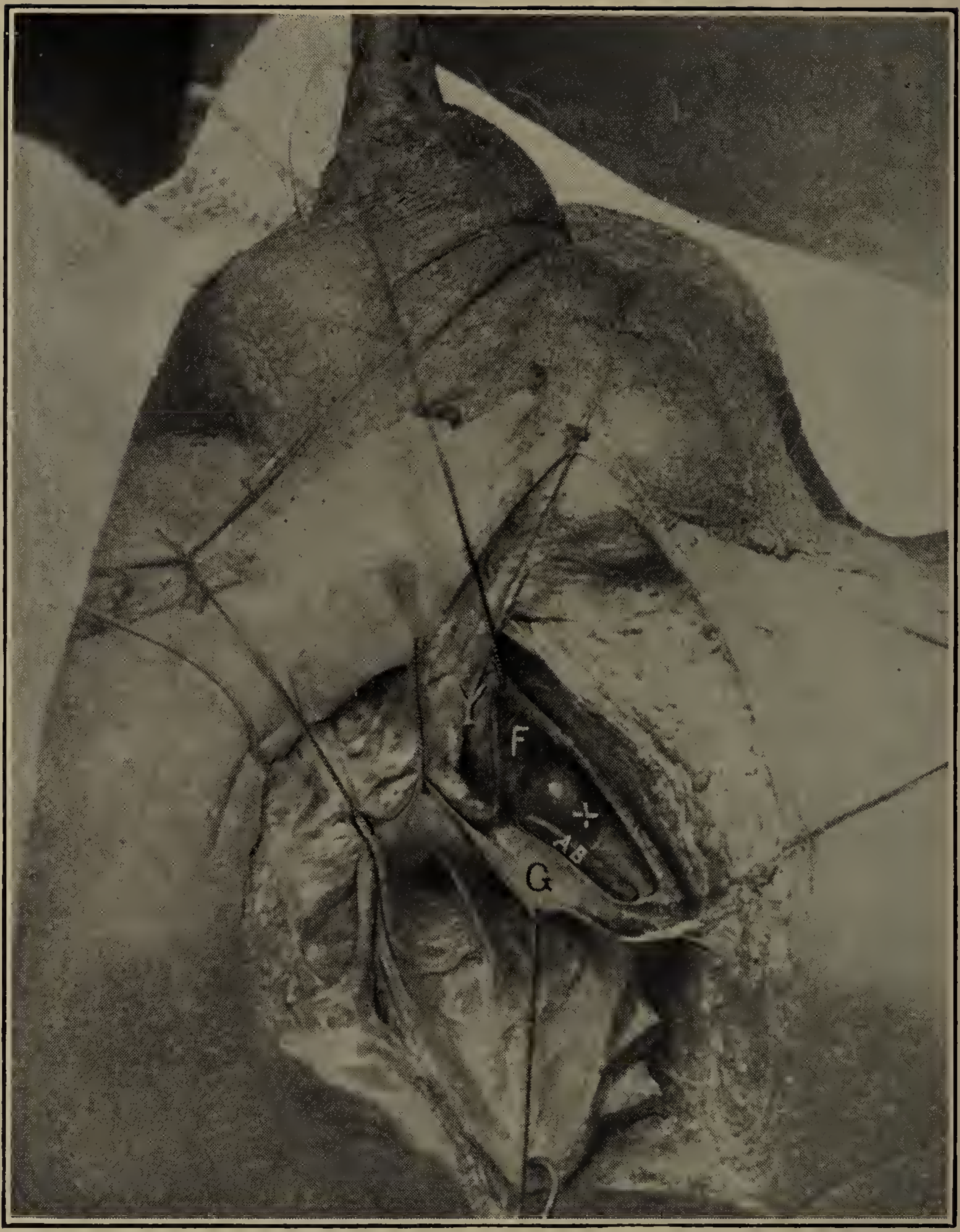

Fig. 8. (photo.) The structures of the anterior urethral triangle have been cut away on the left side down to the superficial layer of the triangular ligament which has been cut through and the flap F reflected, opening up the compartment of the triangular ligament. Within the compartment are seen an oval elevation (+) corresponding to a distorted position of the membranous urethra covered by the constrictor urethrae muscle, which has been projected laterally on the tip of a sound introduced within its calibre ; also the artery to bulb (AB) which lies at the base of the compartment. Y bulbous urethra over sound can be traced in shadow. G lateral half of roof of double compartment, contains transversus perinei muscle encased in fascia.

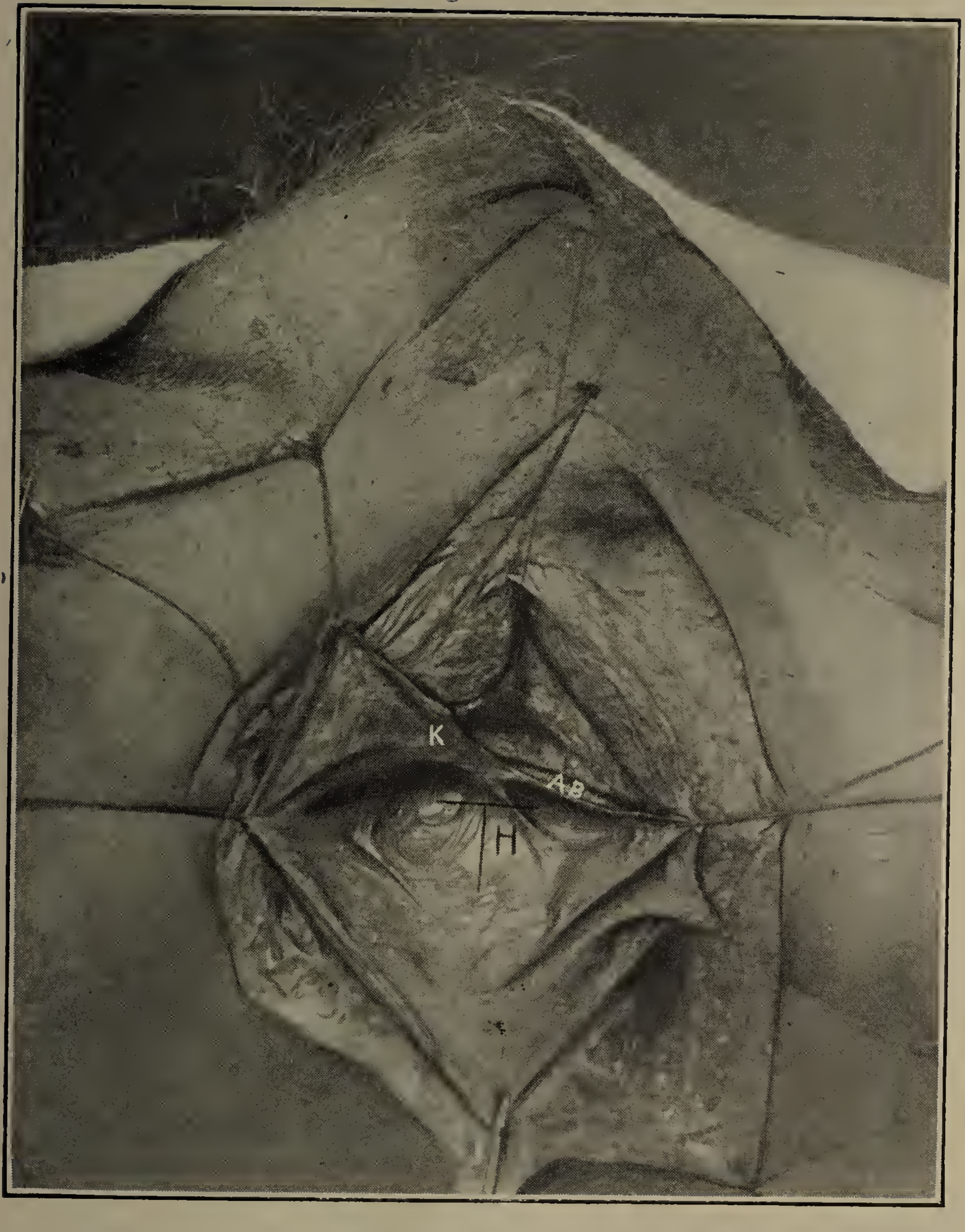

Fig. 9. (photo.) Fibrous vault at base of triangular ligament opened up after division of the median septum of the double compartment (fig. 7) and lateral stripping of the radiating muscle fibres in its floor. The roof of the vault has been split longitudinally on the left side up to the artery of the bulb (AB). K central tendon of perineum. H recto-vesical fascia (fig. 14, H) covering the recto-urethralis muscle (figs. 10 and 14 H′). Transverse black line indicates site of incision through the recto-vesical fascia only, very close to K. Vertical line is the site of longitudinal splitting of the recto-urethralis muscle by blunt force.

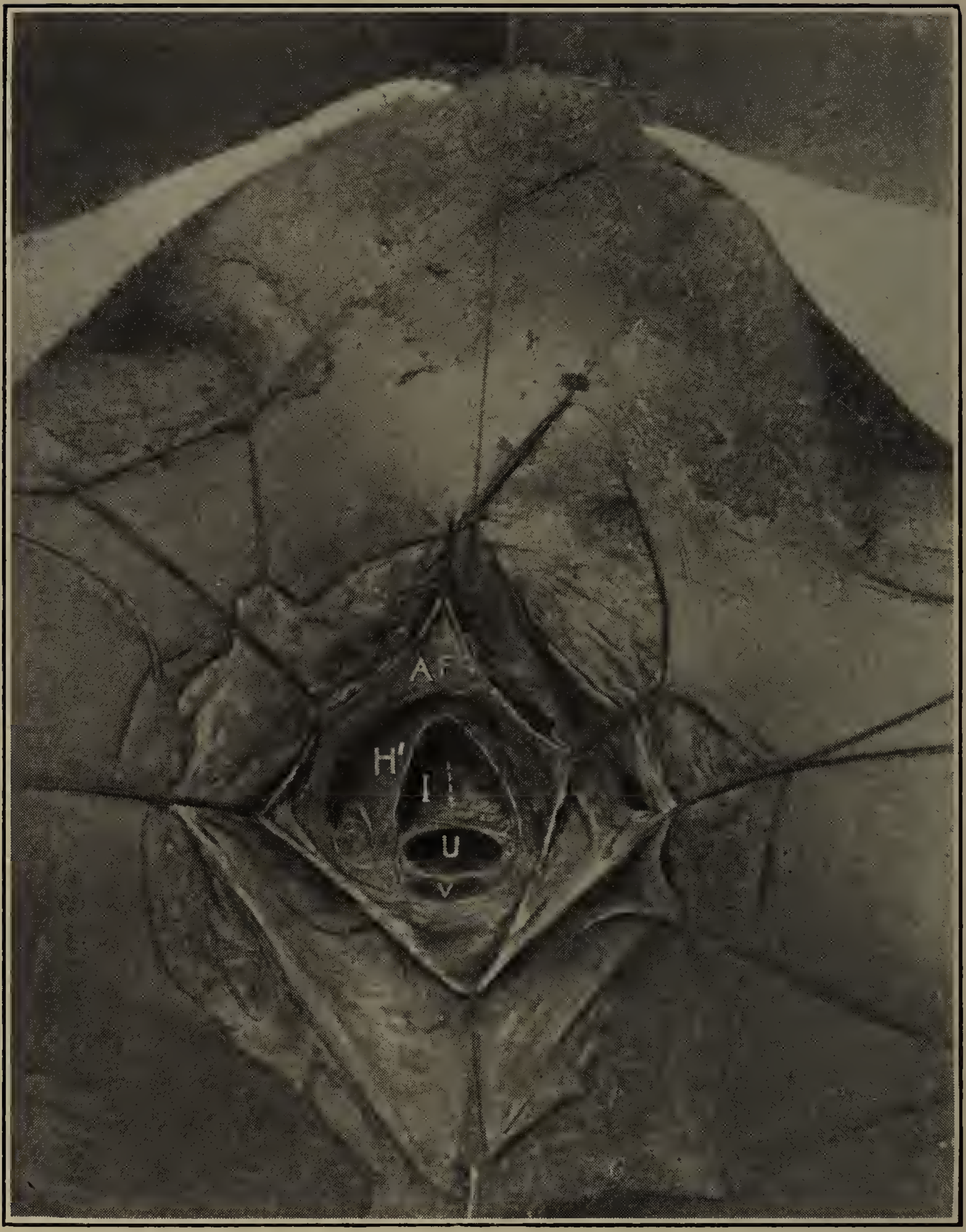

Fig. 10. (photo). The recto-vesical fascia A F (fig. 9, H) has here been cut transversely across and the opening stretched by traction to show the recto-urethralis muscle (H′) beneath, the latter split longitudinally. The rectal wall has been peeled back from the deep aspect of the recto-urethralis muscle, and from the basal layer of the triangular ligament or U-shaped space (I) (cf. fig. 14). V longitudinal coat of rectum here having had a thick fascial covering which has been partly dissected off. U placed in the oval opening of entrance to the fascia-lined infravesical or prerectal space, situated at the apex of the prostate where in this dissection there was no insertion of the longitudinal coat. The dotted line idicates the situation of the membranous urethra above.

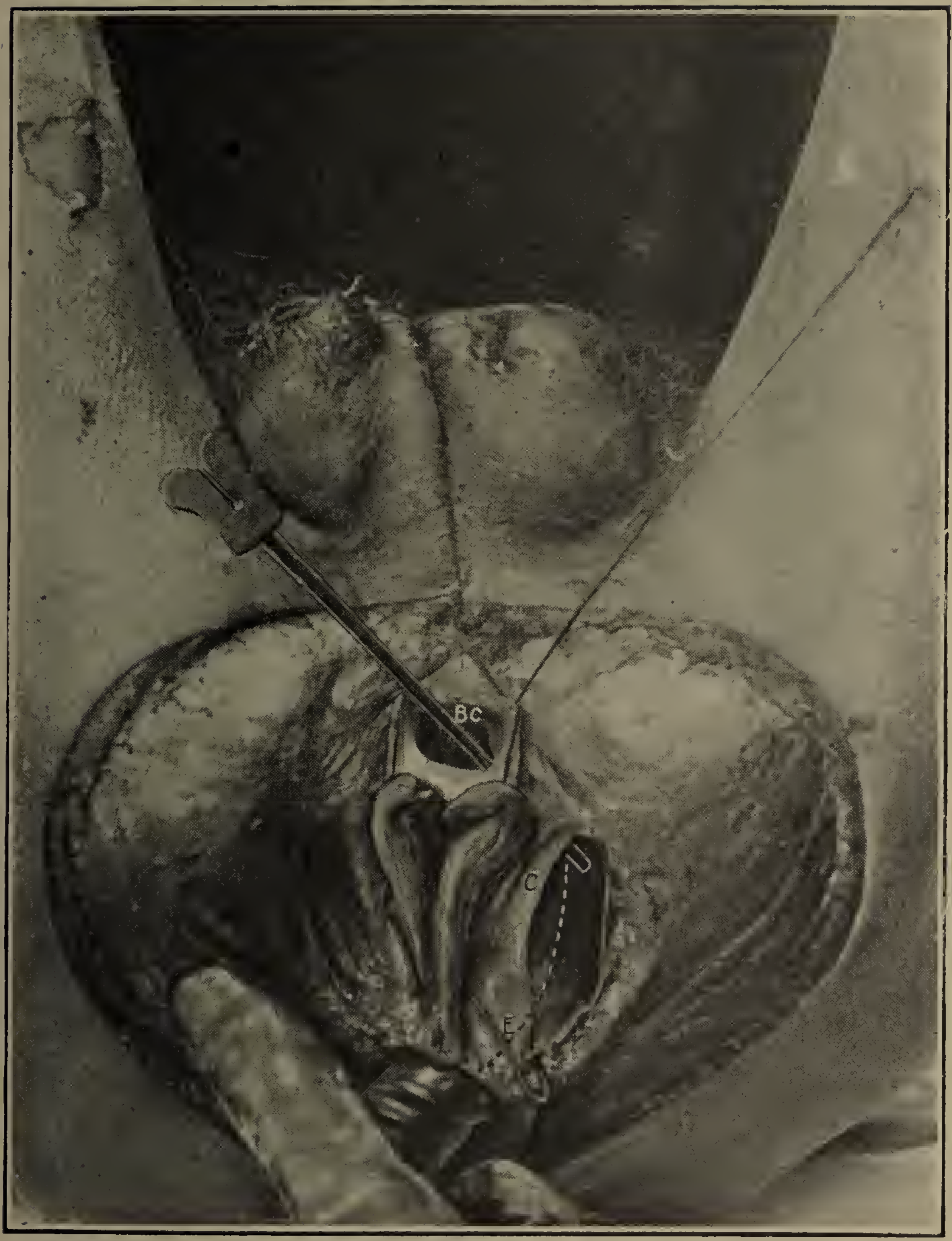

Fig. 11. The grooved director has been inserted in the picture to indicate how the bundles B and C, fig. 3, can be raised antero-laterally from the anterior bundle of the levator ani (fig. 12, M) beneath. The finger has been passed through a lateral incision in the floor of the space from which the coccyx has been removed, entering the postero-lateral fascial compartment (fig. 13, Q). E anal fascia. Dotted line, incision through levator ani.

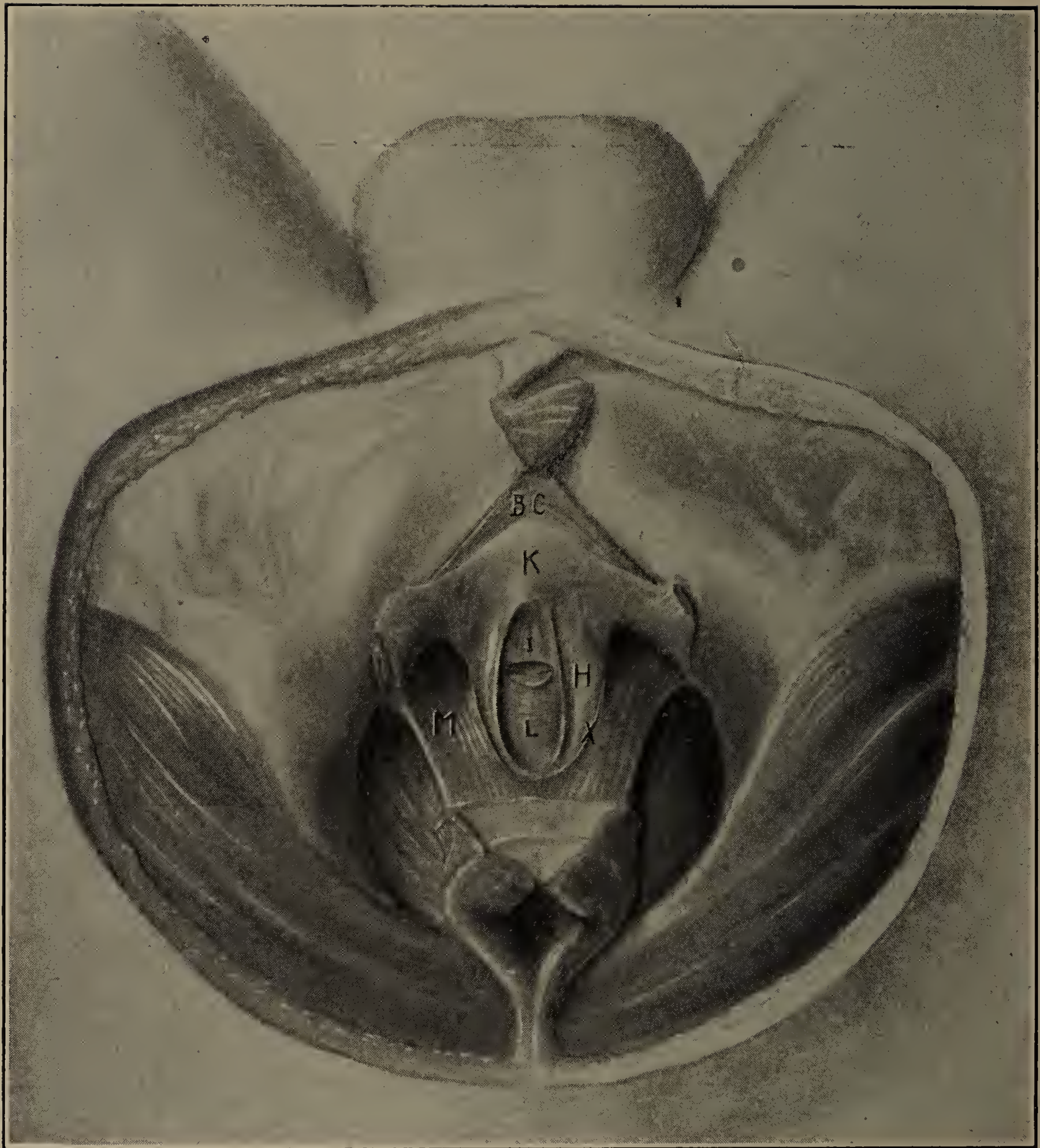

Fig. 12. (drawing semi-diagramatic). Antero-lateral bundles B and C (fig. 11) have been divided and the anterior bundles of levator ani (M) exposed beneath. Compartment between central tendon of perineum (K) and recto-vesical fascia (H) opened up. X muscular margin formed after division of the median septum of the double compartment (fig. 7) and stripping the muscular fibres in the floor of the space laterally. The recto-vesical fascia and recto-urethralis muscle are split open in the median line showing circular coat (L) of rectum separated back a little ways from the under surface of the recto-urethralis muscle and from the basal layer of the deep urethral compartment (I) which latter here receives the insertion of a few fibres of the longitudinal coat. The attachment of the longitudinal coat to the apex of the prostate is cut showing entrance just behind I to the prerectal space at base of bladder. BC deep anterior loop of external sphincter.

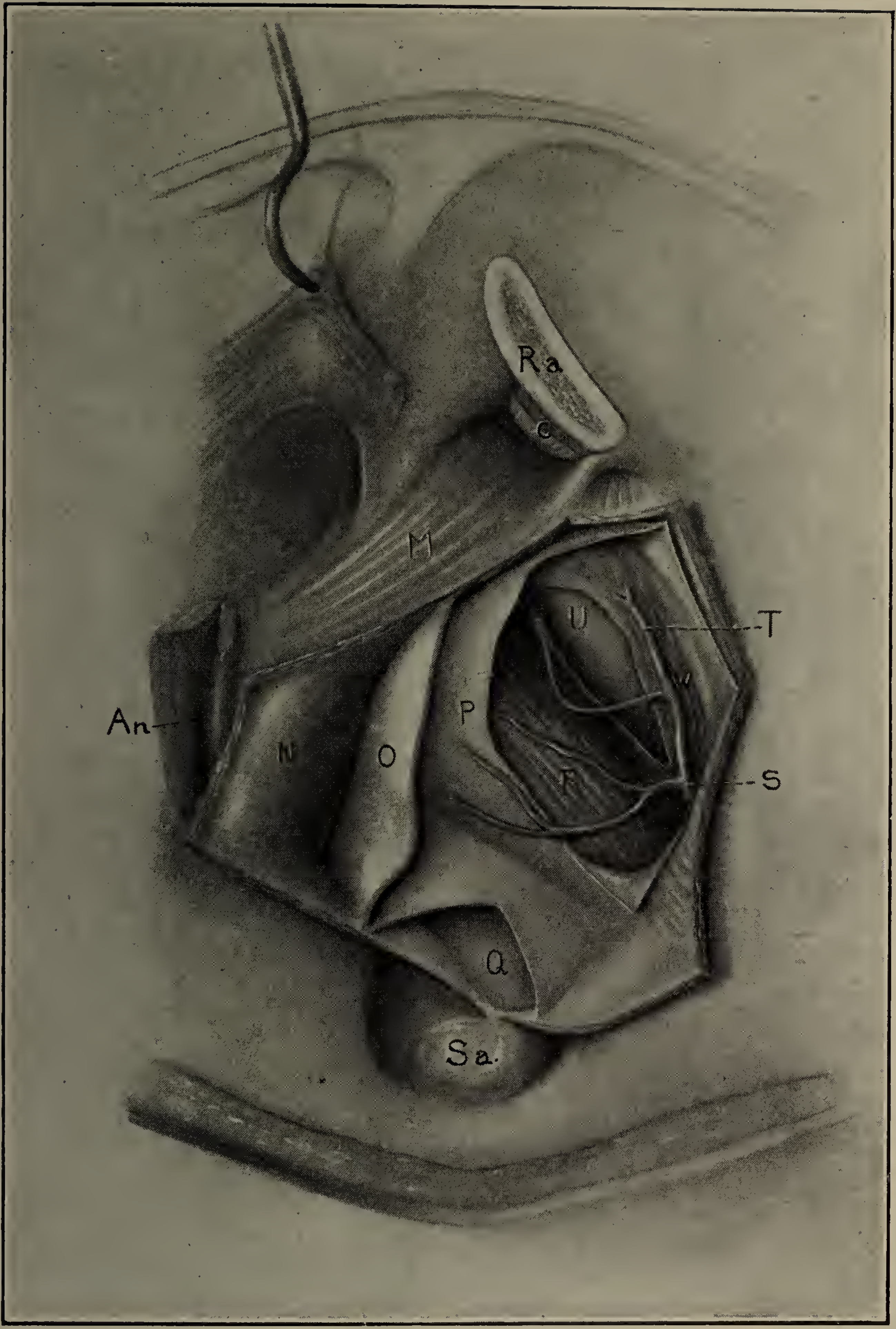

Fig. 13. (Essential parts drawn from photo). Shows lower rectal region. Ramus and tuberosity of ischium cut away to facilitate the deep dissection of the rectum (R). Ra pubic ramus. Sa tip of sacrum. An anus. The triangular ligament has been divided laterally to show the anterior bundle of the levator ani (M) passing above it. N outer layer and O an inner layer of levator ani, reflected. P recto-vesical fascia cut open and reflected from antero-lateral portion of rectum to show middle hemorrhoidal artery (S), its branch to the prostate (T), and the infravesical, or prerectal, space (U) opened up. U is placed on base of prostate, at which situation the artery enters. Q postero-lateral fascial compartment into which the finger introduced through the opening beneath coccyx (fig. II), enters. W fascia covering vesiculis seminalis, C cut end of pubo-rectalis (fig. 3).

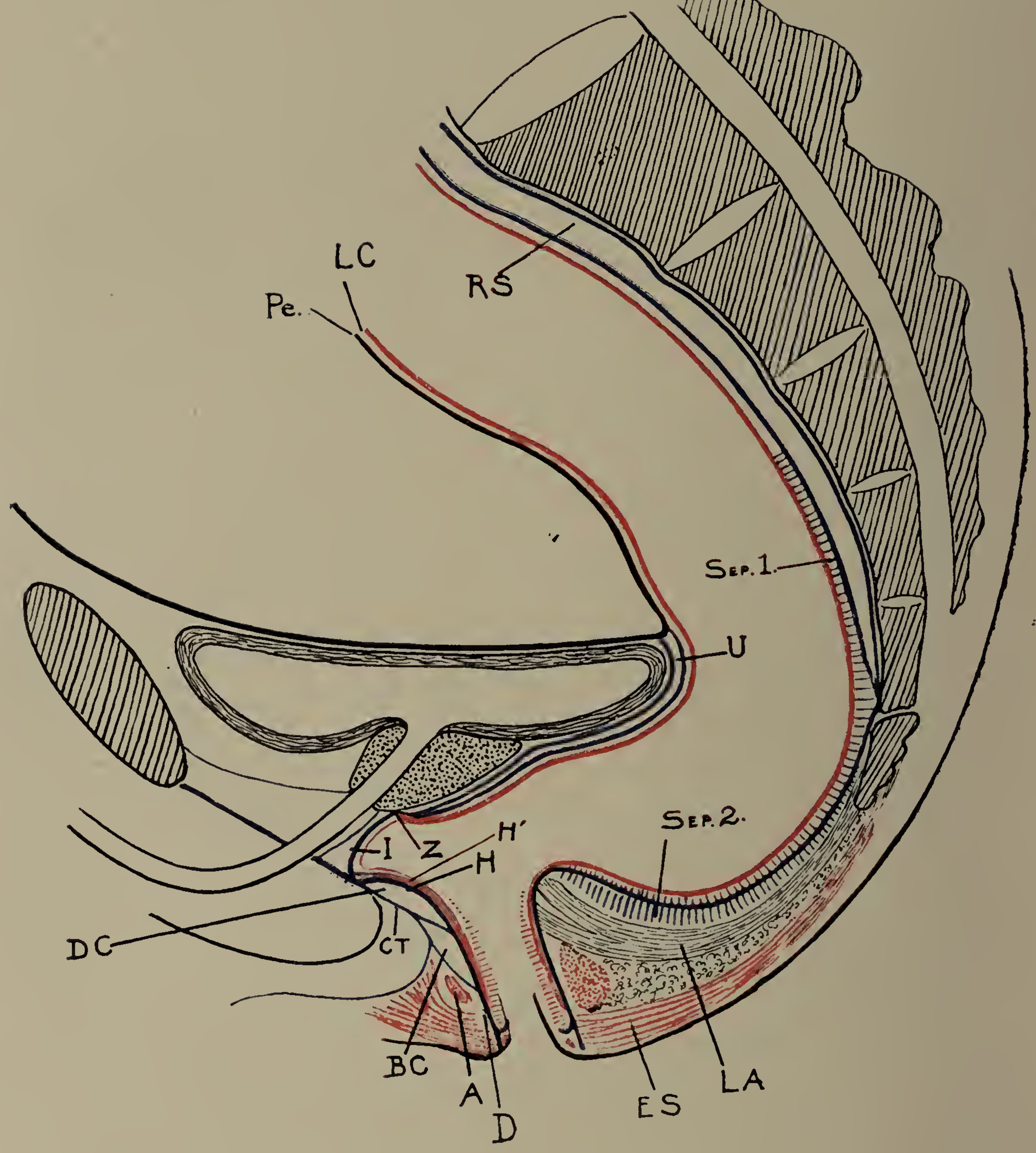

Fig. 14. (Diagram). The fascias are in blue, the muscles in red. ES external sphincter. A superficial anterior loop of external sphincter. BC compartment for deep anterior loop of external sphincter (BC, fig. 6). D aponeurotic layer separating external from internal sphincter, above continuous with recto-vesical fascia which divides into a layer to central tendon of perineum (CT) and a deeper layer (H) covering the recto-urethralis muscle (H'). The fibrous deep surface of the latter is represented by the blue line. DC double compartment with septum, shown in fig. 7. Behind the upper portion of the recto-urethralis muscle is the separable interval between it and the rectal wall, which extends upward beneath the basal layer of the deep urethral compartment (I). Z attachment of longitudinal muscular coat (LC) to apex of prostate, (not constant). The red line continued downward from Z indicates here a thin layer of longitudinal fibres which join below with the recto-urethralis muscle. U infravesical or prerectal space. RS retrorectal space. Sep. 1 posterior median septum attaching the longitudinal coat to the fibrous sheath of the rectum up to the level of the middle of the third sacral vertebra. Sep. 2 median septum between the postero-lateral compartments Q, fig. 13. LA levator ani. Pe peritoneum.

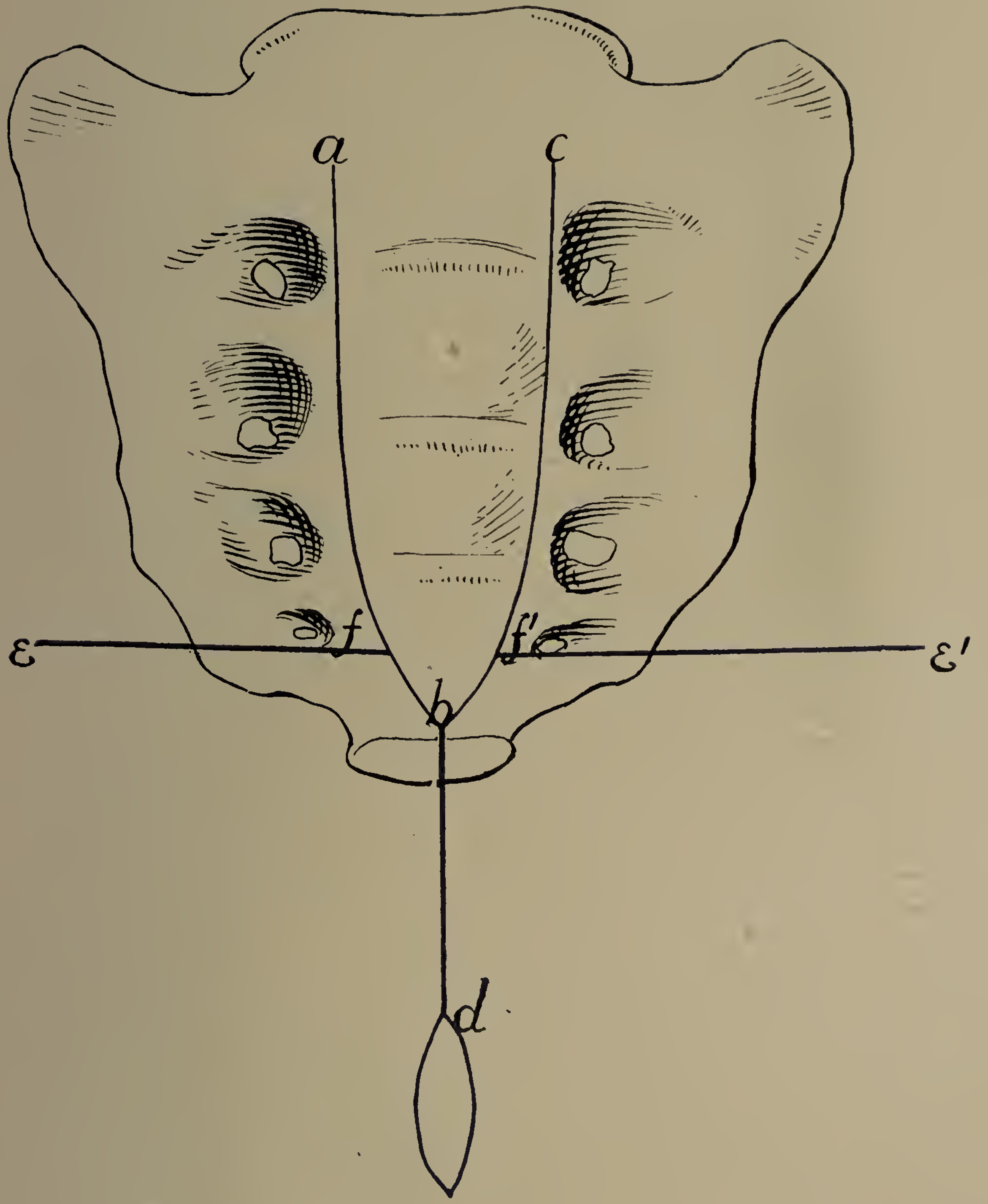

Fig. 15. (Diagram). a b c V-shaped line of attachment of the fascia behind the bowel to the anterior surface of the sacrum, forming the lateral boundaries of the retrorectal space (fig. 14, RS). Ef, e'f' lines of attachment of recto-vesical fascia along upper borders of lesser sacro-sciatic ligaments. bd indicates the continuance downward of the recto-vesical fascia from the lower end of the sacrum to to the anal canal.

THE RATIONAL TREATMENT OF TETANUS: A REPORT OF SUCCESSFUL TREATMENT BY SPINAL SUBARACHNOID INJECTIONS OF ANTITETANIC SERUM, WITH OBSERVATIONS.

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Prior to the discovery of the *bacillus tetani* by Nicholaier in 1884, the treatment of tetanus had been empiric and symptomatic. The cultivation of the bacillus in an anærobic medium by Kitasato in 1891, and the discoveries of Catani, Tizzoni and Behring, were followed by the more rational treatment of tetanus; its serum therapy.

But even this has by no means given entire satisfaction. The subcutaneous injection of the antitetanic serum in 290 cases collected by Moschcowitz gave a mortality of 40.33 per cent. The failure of this method of administering the serum to materially lower the death rate induced Roux and Borrell to make an attempt to bring the serum into more intimate contact with the higher nerve centres, the parts supposed to be mostly affected; thus originated the intracerebral injections. Of 48 cases so treated, collected by Moschcowitz, there was a mortality of 52.08 per cent. This very high mortality is, no doubt, due in part to our rather limited knowledge of the pathological anatomy, and particularly of the pathogenesis of tetanus.

All original observations in this field are by no means concurrent. The most generally accepted view at present points to the motor ganglia of the anterior horns of the spinal cord as being the seat of origin for the tetanic contractions. The more or less constant morphological changes found in these cells of the anterior horns of the spinal cord by Goldschieder and Flatau, both in experimentally tetanized rabbits and in autopsies on patients who died from tetanus, would seem to substantiate this view.

The cerebro-spinal fluid, according to Stintzing, contains a much more active and concentrated toxin than does the blood. He believes that the tetanus bacillus produces toxins at the seat of infection, and that these toxins are carried along the nearest nerves, probably in the interstices of the perineurium and the endoneurium to the spinal cord, and reaching the subarachnoid space of the cord, they produce their toxic effect primarily at the point of entrance into the cord. Lambert's work seems corroborative of this view. Moschcowitz also says "that the point of attack for the toxins is certainly the motor ganglia cells of the anterior horn, which get into a state of increased reflex irritability from the action of the toxins."

Blumenthal and Jacobs made a noteworthy attempt, experimentally, to bring the serum into more intimate contact with the primarily affected parts and the toxins; that is, into the spinal cord and spinal subarachnoid space. They experimented upon tetanized goats, all of which died, the failure probably being due to the small subarachnoid spaces in goats, consequent small amount of fluid, and, therefore, slow endosmosis. They concluded that, by the time the chain of symptoms collectively diagnosed as tetanus had presented, the tetanic poison was so firmly fixed in the central nervous system that it could not be removed, and they furthermore warned against a too hopeful result of the intracerebral method of Roux and Borrell. Jacob, since then, reported two cases of tetanus cured by the injection of the tetanus antitoxin into the spinal subarachnoid space.

Accepting the above rather brief statement as the present view of the pathogenesis of tetanus, and that the primary lesion is in the motor ganglia of the anterior horns, and that the spinal fluid contains more highly active toxins than does the blood, then, by ratiocination, we conclude that the spinal subarachnoid injections of the tetanic antitoxin, preceded by the withdrawal of the active and highly toxic spinal fluid, is indisputably the rational treatment of tetanus. This does not apply to the rare forms of head tetanus.

With this brief *résumé,* we will report the five following cases:

**Case I.*—P. McB., male, twelve years old, admitted to my service at the Harlem Hospital July 2, 1902, with the following history: Six days ago received blank cartridge pistol shot wound in palm of left hand. Was treated in an uptown dispensary, where the wad was presumably removed and the wound thoroughly carbolized with pure carbolic acid. No constitutional symptoms presented until five days after injury, when patient suddenly had a violent attack of dyspnea, followed by slight tremor of left hand and rigidity of muscles of neck and thorax. From the beginning of the attack of dyspnea had constant severe pains in the back and frequent convulsions. Contraction of the muscles of the abdomen and the so-called locking of the jaws were the last symptoms to appear. The period of incubation in this case was evidently five days.

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*Cases I., II., III. were reported in *Medical News* April 18, 1903.

Examination showed a well-developed lad; heart and lungs normal; urine negative. Palm of left hand presented small, round wound, between the first and second metacarpal bones, edges powder-burned. Bottom of wound grayish in color, from which a very small amount of pus could be expressed. No inflammatory reaction surrounded the wound. Accepting Rotter's subdivision of tetanus, would put this case in Subdivision B—a grave case.

Convulsions one after another, and most violent, particularly the muscles of respiration, diaphragm and abdomen. The admission temperature of 98 degrees F., pulse 104, respirations 22, rapidly rose in twenty-four hours to temperature 106 degrees F., pulse 112, respirations 38 (pulse and respirations counted between the convulsions), and the patient died in the most violent tetanic spasms, particularly of the muscles of the chest. In fact, a member of the House Staff remarked that these contractions were so strong that they simply "squeezed the life out of him." This expression is also used in Allbutt's "System of Medicine," and is not to be wondered at, since they have been so strong as to cause a rupture of the rectus abdominalis and psoas magnus. Indeed, a case has been reported in which both femurs were fractured by the force of contracting muscles in a case of tetanus, and another in which the second cervical vertebra was dislocated. The diagnosis in this case was confirmed by Dr. Harlow Brooks, pathologist to the hospital, who succeeded in finding a very few tetanus bacilli in a culture taken from the wound.

Treatment.—Wound freely opened and swabbed out repeatedly with pure carbolic acid. Wet dressing applied. Bromides and chloral freely administered. Forced nutrition and stimulation.

Hot packs and prolonged hot baths. In fact, he received the useless, empiric and symptomatic therapy usually exhibited in such cases.

This case is reported in contrast to Cases II., III., IV. and V., and in order to emphasize a protest against the useless and sometimes reckless and dangerous employment of potassium bromide and particularly of chloral hydrate, especially against their continued use in cases of tetanus when it is clearly seen that they are absolutely of no avail. They may alleviate the distress, but they do not modify the progress of the disease.

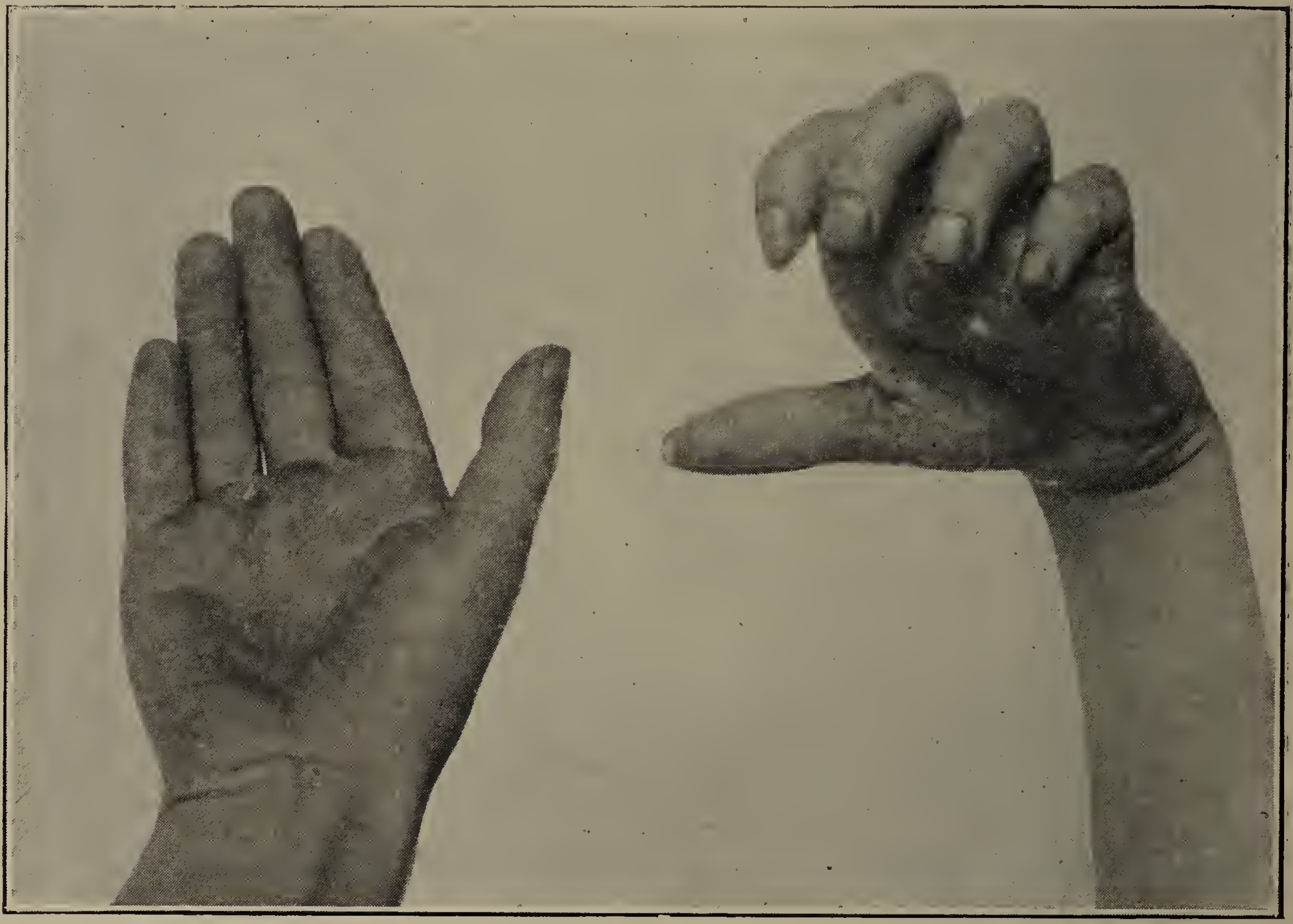

Fig. 1. Case II.—Tetanus—wound, left palm between 2d and 3d metacarpi. The claw-like contraction of fingers is known as "local" contraction of tetanus and is rarely observed in tetanus, for when present it usually disappears before case is seen by surgeon. It is not due to an infectious cellulitis nor synonitis.

Case II.—Male, twelve years old; admitted to my service at the Harlem Hospital July 12, 1902. Family and personal history negative. Previous history: Diphtheria and scarlet fever six years ago. He received a blank cartridge pistol shot wound in palm of left hand July 4. The wound had been cleaned and a wet dressing applied at an uptown dispensary. No constitutional symptoms developed until the seventh day, when the child complained of stiffness in the jaw, pain in the back of the neck and right side of chest. Admitted to the hospital on the following day.

Examination.—Well-developed lad, with heart and lungs normal. Small, round wound in the centre of palm of left hand, midway between the second and third metacarpal bones, grayish in color. Edges powder burned. Discharge scant. Most noticeable feature about this hand was the claw-like contraction of all the digits except the thumb, which was in a state of hyperextension, and flexion of wrist on the forearm. This condition was not due to any inflammatory changes (see Fig. 1). There was no cellulitis nor tenosynovitis present. It was due, however, to the so-called local effects of the poison, and is not ordinarily observed in cases of tetanus. If present at all, it does not usually last long after its first manifestation, and it thus frequently escapes observation, but in this case it continued for two weeks. Jaws firmly set; risus sardonicus present. Muscles of the abdomen in a state of constant tonic contraction. There was also present in this case marked tonic contraction of the orbicularis palpebrarum, the sphincter muscle of the eyelids (see Fig. 2, from photograph taken on the fifth day after injection). Although the patient is making a great effort to open both eyes and mouth, the left eye is only partially open, the mouth only slightly so. The jaws had somewhat relaxed when photographed. Notice, also, the contraction of the muscles of the neck. They are very rigid, but no opisthotonos present. The shoulders are raised from the bed by the nurse's hand placed at back of head.

When admitted his temperature was 101 degrees F., pulse 116, and respirations 24. The highest temperature, reached on the third day, 104 degrees F., became normal on the tenth day. Frequent examination of pus by Dr. Brooks failed to reveal the *bacillus tetani*, but the clinical diagnosis, however, was undoubtedly that of tetanus.

Treatment.—In order to give the serum therapy as fair a chance as possible, strict orders were given that no other treatment was to be employed; but, due to a misunderstanding, the wound was swabbed out with pure carbolic acid, once only, which probably partly accounts for the failure to discover the bacillus. Under careful asepsis, a lumbar puncture was made between the third and fourth lumbar vertebræ, 20 minims of the cerebro-spinal fluid were withdrawn, and 8 c. cm. of the antitetanic serum were injected into the spinal subarachnoidean space. The injection was made very slowly, consuming about five minutes in time to empty the syringe. There were absolutely no ill effects. On the contrary, after a few minutes the tonic contractions all over the body diminished, and were followed by rest and sleep. Pains in the back of the neck ceased. Slight relaxation of the muscles of the jaw, but not enough for mastication, or even to drink from a cup. This remission of the tonic contractions and convulsions lasted about six hours, when the spasms again set in and the patient had intense headaches. In another hour the pains were as severe as preceding the injection, especially in the dorsal region and in the abdomen, which was so contracted as to present the boat shape

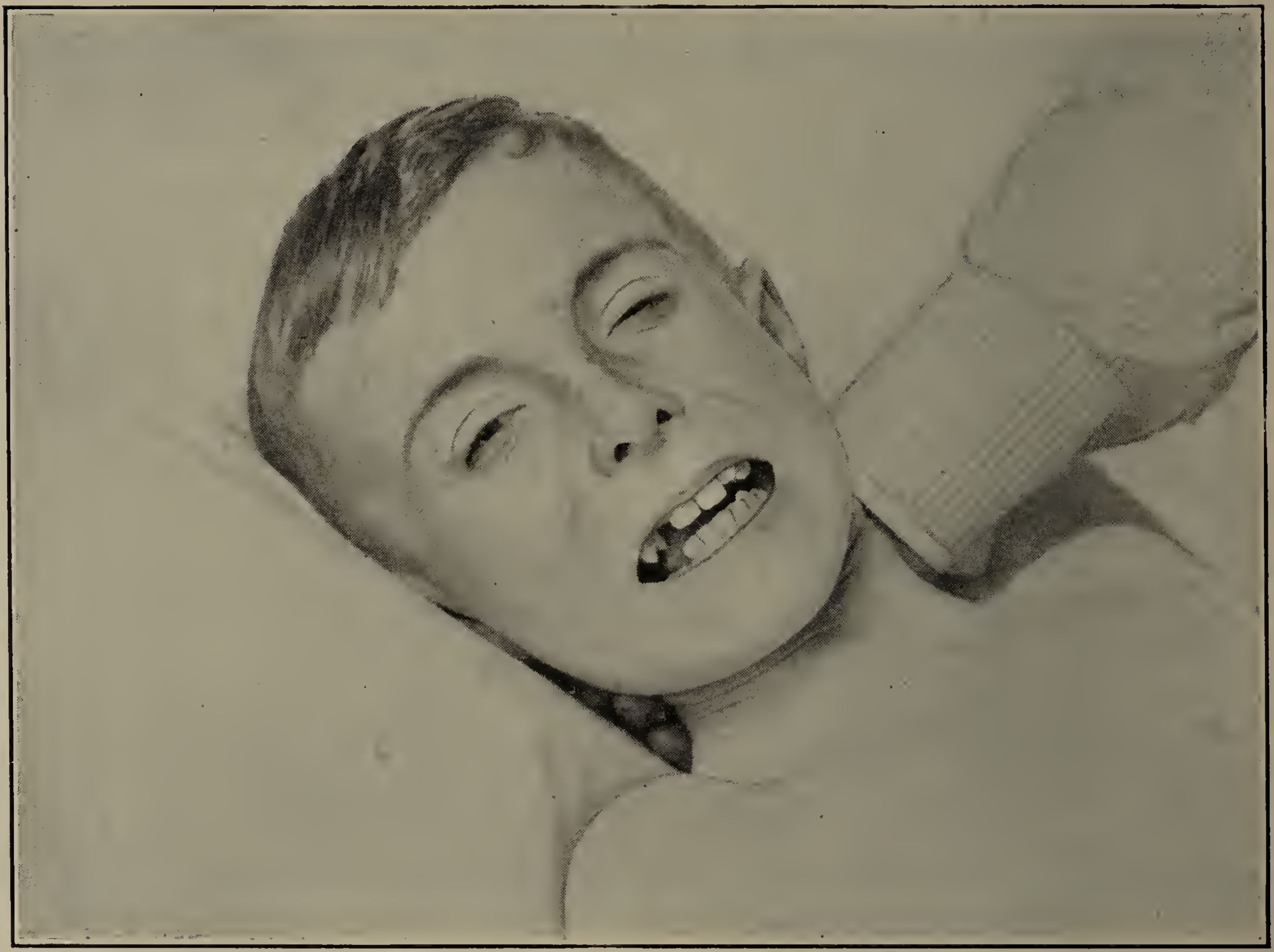

Fig. 2. Case II.—Tetanus—showing tetanic contraction of cervical muscles, muscles of the jaw and the orbicularis palpebrarum (left eye nearly closed). Antitoxin rash on face. Teeth extracted to insert feeding tube. Photographed on fifth day after injection of antitoxin.

of cerebro-spinal meningitis. This would last less than five minutes, when it would partially relax. On the following day, July 13, another lumbar puncture was made, 12 minims of spinal fluid withdrawn and 11 c. cm. of serum injected, followed by rest, remission of contractions, less pain, etc., as after the first injection. In fact, the patient received daily spinal subarachnoid injections for eight days, all between the third and fourth lumbar vertebræ, as follows:

July 12—22 drops of cerebro-spinal fluid withdrawn; 8 c. cm. tetanus antitoxin injected.

July 13—12 drops cerebro-spinal fluid withdrawn; 11 c. cm. tetanus antitoxin injected.

July 14—5 drops cerebro-spinal fluid withdrawn; 12 c. cm. tetanus antitoxin injected.

July 15—40 drops cerebro-spinal fluid withdrawn; 10 c. cm. tetanus antitoxin injected.

July 16—2 drops cerebro-spinal fluid withdrawn; 12 c. cm. tetanus antitoxin injected.

July 18—None withdrawn; 12 c. cm. tetanus antitoxin injected.

July 19—35 drops cerebro-spinal fluid withdrawn; 12 c. cm. tetanus antitoxin injected.

July 24—45 drops cerebro-spinal fluid withdrawn; 15 c. cm. tetanus antitoxin injected.

—making a total of 161 drops of cerebro-spinal fluid withdrawn and a total of 92 c. cm. of the tetanus antitoxin injected.

On July 26 the patient sat up in bed for two hours; was out of bed on July 28, and on July 29 could open mouth as normal; complete relaxation of all muscles. Was discharged from the hospital, cured, on July 30.

Case III.—W. B., male, ten years old. Family and previous history negative. Admitted to the Harlem Hospital September 10, 1902, on the service of Dr. Haubold, who kindly permits me to report the case, as it was treated according to our observations in Case II.

Present History.—Seven days ago, while attempting to vault the garden fence, fell and cut left wrist on the neck of a broken bottle. Was treated at an uptown dispensary, where several sutures were taken in the wound and a wet dressing applied. Five days after injury had severe abdominal pains. Applied to an uptown hospital for admission and was refused. Was seen on the night of the sixth day after injury by Dr. Bivings, House Surgeon of the hospital, who diagnosed the case as tetanus, and injected 12 c. cm. of tetanus antitoxin, after withdrawing 125 drops of the cerebro-spinal fluid. As the tonic contractions were most marked in the muscles of the abdomen, I believe from the history that the period of incubation was five days, as it was on the fifth day after injury that the patient first complained of the severe abdominal pains which were undoubtedly caused by tetanic contractions. The symptoms in this case, like Case I., were those of Rotter's Subdivision B—a grave case. Without going into further details this case received spinal subarachnoid injections as follows:

September 9—125 drops of cerebro-spinal fluid withdrawn; 12 c. cm. tetanus antitoxin injected.

September 10—100 drops of cerebro-spinal fluid withdrawn; 9 c. cm. tetanus antitoxin injected.

September 11—125 drops of cerebro-spinal fluid withdrawn; 14 c. cm. tetanus antitoxin injected.

September 13—115 drops of cerebro-spinal fluid withdrawn; 12 c. cm. of tetanus antitoxin injected.

September 15—140 drops of cerebro-spinal fluid withdrawn; 12 c. cm. tetanus antitoxin injected.

—making a total of 605 drops of cerebro-spinal fluid withdrawn and a total of 59 c. cm. of tetanus antitoxin injected.

The reaction after each treatment in this case was more positive than in Case II., followed immediately by a cessation of convulsions, relaxation of contracted muscles, etc. The patient sat up in bed September 22 and was discharged, cured, September 26.

Prognosis in this case was, if possible, more grave than in Case II., yet recovery was most satisfactory under injections of only 59 c. cm. of the antitetanic serum, 33 c. cm. less than in Case II. But we withdrew 444 drops more of the highly toxic spinal fluid than in Case II. In short, I believe that the indication is to withdraw as much of the highly toxic spinal fluid as possible.

Case IV.—Female, aged seventeen. Admitted to my service at the Harlem Hospital July 12, 1903, with the following history:

July 4 accidentally shot herself in the palmar surface of the left hand, in mid-metacarpal region, with blank cartridge. Applied at the outdoor department for relief on the same day, but absolutely refused to allow surgeon in charge to open up and curette wound; hence wad was but imperfectly removed. The wound, however, was cauterized with pure carbolic, and an antitetanic powder (which was being experimentally used) was dusted into wound. The patient also refused the injection of antitetanic serum, which procedure was used in all other cases as a prophylactic measure. She appeared at the outdoor department on following day for dressing, apparently in good health.

She was not seen again until July 12, at 11.30 P. M., eight days after injury, when the ambulance surgeon found her suffering with severe pains in head and body, great difficulty in breathing, inability to articulate, with muscular twitchings, locking of jaws and complaining of feeling "just like a piece of lead." She was removed to the hospital.

Physical examination: Absolute locking of jaws; jaw clonus present; retraction of head and marked rigidity of muscles of back of neck, which were also extremely sensitive and tender to touch; marked risus sardonicus; intercostals were very rigid, of wood-like hardness, with marked interference in the respiratory movements of chest wall; respirations also very painful—patient would cry out with pain on deep inspiration or forced expiration.

Abdominal muscles as hard as a board and tender to the touch. Left hand showed the same claw-like contraction of digits, which could only with difficulty be forcibly extended—voluntary extension lost.

Legs were extremely rigid—both patellar reflexes exaggerated. Opisthotonos so very marked that patient actually rested on her head and heels. Convulsive twitching of muscles of entire body, lasting at times for ten minutes.

July 13, condition practically the same as on admission. During the day patient had eighteen convulsive attacks of the muscles of the body, at intervals of twenty to thirty minutes, each attack lasting from three to five minutes. Ten were of the entire body; four of the head and limbs, muscles of the trunk remaining perfectly quiet, and four were of the limbs only.

Two upper incisor teeth were withdrawn, and forced feeding and stimulation began immediately.

Patient received spinal subarachnoid injection, as in Cases II. and III. July 14, patient had but two attacks of muscular twitchings; these were of the limbs only, but she complained of very severe pains over the entire body and most severe in the abdomen. During the day developed incontinence of urine and feces. Slept greater part of night, except when awakened by a sudden sharp pain in abdomen. Temperature, 99.6 degrees F.; respirations, 90; pulse, 26. Spinal fluid withdrawn and subarachnoid injection given.

July 15, markedly improved; had but one convulsion during entire day, this involving head and limbs. Could open mouth ½ inch; complained of very little pain; took nourishment well. Rigidity of muscles of lower extremity somewhat diminished. Spinal fluid withdrawn and subarachnoid injection given.

July 16, patient had but one attack of convulsive twitching of the muscles of the limbs, followed by marked rigidity of all the muscles of the body and some increase in the locking of the jaw, but within two hours after the injection the patient could again open the jaws.

July 17, complained less of pain than on any previous day. Muscular rigidity markedly diminished; no convulsions; opisthotonos nearly gone; general condition good; had not lost flesh.

July 18, general condition good; no convulsions; slept seven hours; nearly total absence of muscular rigidity (although a sudden noise would cause the patient to start, the body would become rigid for several seconds and then gradually relax); incontinence of urine at times. Improvement positive and rapid from this time on.

July 22, patient sat up, out of bed, for two hours. July 25, she essayed to walk, but had difficulty on account of stiffness of all the muscles of back and legs. No longer incontinent. Discharged August 12, cured.

Summary of treatment:

July 13—250 drops cerebro-spinal fluid withdrawn; 18 c. cm. tetanus antitoxin injected.

July 14—491 drops cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.

*July 15—515 drops cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.

July 17—300 drops cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.

Total amount of cerebro-spinal fluid withdrawn, 1,556 drops (oz. iii.+).

Total amount of tetanus antitoxin injected, 78 c. c.

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*NOTE.—317 drops were clear spinal fluid, followed by 124 drops of serum tinged with blood; then 6 drops of clear serum, after which fluid became quite bloody again for 64 drops. I have been at a loss for a solution of this phenomenon, for at no time after the fluid started to escape *was the needle touched, moved or shifted in any manner.*

Frequent examinations of the pus from wound in hand were made, but at no time was the *bacillus tetani* found. This fact was probably due to the thorough cleansing and cauterizing of the wound by carbolic acid and tincture of iodine.

Case V.—Male, twenty-four years old, admitted to my service at the Harlem Hospital, July 18, 1903, with the following brief history:

Accidentally shot himself with a pistol, blank cartridge, in palm of left hand, at the base of the ring finger, on July 4. The wound was absolutely neglected by the patient.

July 14, ten days after the accident, the patient began to have severe pain in the jaws, followed by stiffness and by pain in the back and legs. This is the first case of tetanus that I have seen in which the locking of the jaws was the first clear, distinct and prominent symptom of the impending calamity. Patient rapidly grew worse until, on July 18, he began to have most violent and severe convulsions. The abdominal muscles were hard and board-like—in fact, there were present all the symptoms of a very grave case of tetanus. Although this was the fourteenth day after the accident, it was only the fourth day after the appearance of the first symptoms of tetanus, so I do not consider this a so-called chronic case; nor was it a mild one; nor could a better prognosis be given on account of the time elapsed from the infection to the first symptoms of tetanus, according to Rotter. I would rather call the case one of the *delayed acute* type.

The tetanic convulsions were the most violent of any that I have ever seen, including some cases that I have seen die.

On the second day of injection, while the needle was being inserted, the patient suddenly had one of these violent seizures. I essayed to rapidly withdraw the needle, as the patient was turning over on his back. I was somewhat surprised to be unable to budge the needle—so firmly was it held by the contracted muscles that it could not possibly be farther inserted nor extracted until the convulsion was over and the muscles relaxed. The wound was nearly healed, but it was reopened wide and curetted, pieces of wad removed, and the wound cauterized with pure carbolic acid.

Without going into further details of the history, I will tabulate the treatment:

> July 18—250 drops cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.
>
> July 19—960 drops (oz. ii.) cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.
>
> July 20—1,440 drops (oz. iii.) cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.
>
> July 26—960 drops (oz. ii.) cerebro-spinal fluid withdrawn; 20 c. cm. tetanus antitoxin injected.
>
> Total amount of cerebro-spinal fluid withdrawn, 3,610 drops (oz. vii.+).
>
> Total amount of tetanus antitoxin injected, 80 c. c.

July 29, patient sat up in bed. Discharged, cured, on August 3.

In tetanus there is a marked, most rapid wasting and exhaustion. This wearing out of the physical powers, this body weakness, seems to constitute an important and very grave feature of the disease, which, directly or indirectly, seems to aid in producing a fatal ending. Now, the processes of alimentary absorption, assimilation and elimination apparently are not in the least interrupted nor interfered with. The involuntary muscles of the stomach and intestines apparently are not subjected to the same violent tetanic contractions and spasms as are the voluntary muscles. *The indication, therefore, is very strong and positive for urgent and forced nutrition and stimulation.* All four cases weighed nearly as much upon discharge as they did on admission. I wish to emphasize this part of the treatment, as I consider it of as great importance as the injection of the antitoxin.

Cases II., III., IV. and V. each received daily from 75 oz. to 136 oz. of fluids or other food by mouth, per tube passed through space created by extracting teeth, or through nose, or per rectum, of milk, gruel, beef juice, orange juice, rice cooked until it could be sucked through tube, and bananas, cut into very thin slices and forced between the teeth.

The antitoxin used in the cases was that of the Board of Health of New York, the immunizing power of which is at least one to fifty million, and twenty-two consecutive spinal subarachnoid injections without an infection or complication of any kind bespeaks a perfectly sterilized product, and demonstrates the safety of this method of treatment in trained hands. The New York Board of Health antitetanic serum is not a regular one, at least in appearance, color, consistency and etc. I have made futile efforts to ascertain the cause of this great variation, as well as to get a description from the Department that would enable one to recognize a standard product.

About every third or fourth bottle is of a different color, varying from champagne to dark amber. Some specimens have been very pale, actually effervescing, and when poured into a glass or drawn into the barrel of a syringe it has been absolutely impossible to expel the little gas or air bubbles that form and stick to the sides of the syringe or glass. These bottles of effesvescing serum I have regularly discarded as dangerous on account of the harmful or fatal effect, problematical or actual, of injecting air or gas into the cord or its spaces.

The best results follow the injection of the dark amber colored serum. This specimen is perfectly free from gas or air, and is decidedly sticky and viscid.

In the course of the observation and treatment of these cases I came to the following conclusions:

1. As no attempt was made to maintain an equation between the amount of spinal fluid withdrawn and the amount of serum injected, that the shock or collapse so often observed following spinal subarachnoid injection of cocaine for anæsthesia is not due to an upsetting of the equilibrium of the pressure in the spinal canal, as is claimed by the advocates of this method of producing anæsthesia, but is due solely to the physiological action of the cocaine.

*2. Given a wound infected with tetanus, I am by no means positive that the anatomical seat of the wound does not play an important röle in the production of the constitutional symptoms of tetanus; for, again accepting the present view of the pathogenesis of tetanus, any wound infected with tetanus near a large nerve, particularly if the nerve sheath be opened, will more likely be followed by constitutional effects of the infection than a wound anatomically remotely removed from the seat of a large nerve. It is possible, therefore, that the space in the palm of the hand

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*The layman has long insisted that the above-mentioned space is a favorite point of infection for tetanus; as a child, I can well remember the fear and consternation of everyone occasioned by a wound in the palm for fear of tetanus. The text-books and all writers have hitherto denied this.

between the first and second metacarpal bone *is a favorable point for an infection of tetanus to produce constitutional effects,* because the median nerve in the hand becomes flattened out and much larger at this point; it therefore offers an increased surface, with greater facilities for gathering and conveying the toxins to the cord.

3. While I recognize the absurdity of trying to draw conclusions from four cases, the reaction after each injection was so prompt, and the improvement so positive, and followed by final cure in four grave cases, that I am constrained to state my belief that in the *spinal subarachnoid injections of the tetanus antitoxin preceded by the withdrawal of the active, concentrated, highly toxic spinal fluid, supplemented by forced nutrition and stimulation, and proper care of the wound,* we have at least the promise of a method of treatment of tetanus by means of which we can offer more hope in this fatal and rightly most dreaded disease than we have been able to heretofore.

CYSTOSCOPIC DIAGNOSIS IN VESICAL AND RENAL SURGERY.

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The degree of importance that the cystoscope, with its modern improvements, has attained in the diagnosis of bladder and kidney affections, is not yet generally appreciated.

A review of some of the more recent work which has been done by the aid of the cystoscope, with a résumé of those conditions in which cystoscopy is especially indicated in its relationship to surgery, will be of interest to those seeing this class of cases.

It is not intended to demonstrate the virtues of cystoscopy as a method to replace the longer established means of diagnosing diseases of these organs, but to show what it has added to these means in bringing to light those conditions of the urinary tract so difficult of diagnosis.

The Bladder is the seat of a number of well-known pathological conditions in the early and accurate diagnosis of which the cystoscope plays the most important rôle.

Tumors.—Of all vesical conditions there are none which depend so absolutely upon the cystoscope for diagnosis as neoplasms. The symptoms, subjective and objective, though suggestive, are never sufficient to leave upon the mind an image of the real condition. It is when the new growth is actually seen, and its site, its size, its form, its character in detail; whether one or two or more; if possible, whether sessile or by a pedicle attached, are noted, that we are in a position to suggest a diagnosis. There are but a few conditions that stand in the way of successful cystoscopy in these cases; *i. e.*, where the urethra cannot be made to receive a cystoscope, where the bladder is entirely filled with new growth, where the transparency of the medium introduced cannot be kept momentarily clear and in advanced cases of malignant disease, which cause unusual irritability or contraction of the bladder. When the patient is seen early enough most of these difficulties are swept away.

Stones.—It is said that Guyon, of Paris, is able to determine the size, the character, and sometimes the number of calculi in the bladder by means of the metal searcher. The opportunities are certainly not given to surgeons of this part of the country to attain such skill, nor is it necessary, for the cystoscope can be used with as little risk and as little discomfort to the patient as the stone searcher, and with an infinitely more satisfactory result. The exact position of the stone is learned, and whether it be encysted or free; the number of stones, if not too many, is ascertained; and what is more, the state of the bladder wall and existing complications are noted. After litholapaxy it is especially important to *see* whether all the particles of stone have been gotten rid of, for if a small fragment remains it will serve as a nucleus for new calculus formation.

Prostatic Hypertrophy.—There is a certain number of cases where the urethral distance or tortuosity, where the cystitis or the easily excited hæmorrhage puts cystoscopy out of the question. In these cases a correct estimation of the extent and character of intra-vesical enlargement or vesical complications, as tumor, stone, etc., cannot be made, and, therefore, we cannot expect to institute such effective treatment nor give by surgical intervention such uniform results as in the other and fortunately larger class, where by the cystoscope we learn the vesical aspect of the prostate, the internal orifice, the *bas fond*, and the condition of the bladder, its mucous membrane, its musculature, its ureters and their doings; and with this knowledge proceed to treatment.

Foreign Bodies such as pins, pieces of catheter, silk sutures, etc., are met with at rare intervals in the bladder, and are generally very easily identified.

Other vesical conditions which are studied by the cystoscope, such as cystitis, vesical tuberculosis, ulcers, varicosities, diverticula, vesicles and bulbous œdema, will not be considered in this paper, as they do not come under the head of those affections ordinarily requiring surgical intervention.

The Kidneys.—Through the agency of the cystoscope and ureteral catheterization it has become possible to make the diagnosis in cases where the most careful observation of all clinical signs leaves the real condition undetermined. But besides diagnosis, perhaps the most important realm of usefulness lies in the field upon which we are just entering, where a true estimate of the functional activity and ability of the separate kidneys is sought. And here is added a new responsibility for all those doing kidney work, and an obligation to every patient of this class; for to remove the kidney on one side without having previously attempted to determine the functionating power of the other gland, which is destined to perform the entire work of renal elimination for the body, is surgery which should be no more.

As to Diagnosis.—Before the day of the cystoscope the observation of the clinical picture, with its characteristic subjective symptoms, and the exact examination of the urine, with the objective knowledge gained through palpation and percussion, were the means we had at hand to solve the difficult diagnostic problems of the urinary system. The value of these means is not contended, but in many cases they are insufficient. To show how misleading are subjective symptoms: There may be pain in the left kidney when the right is the seat of disease. That pain which is characteristic of the presence of bladder or kidney stone may be entirely absent. Great urgency of micturition is most often due to trouble about the vesical sphincter or prostatic urethra, yet pyelitis and renal calculus may cause this same frequency. With pain in the glans penis the trouble may be either in the bladder or the kidney. Pain may come in attacks, as is ordinary with kidney stone; yet pyelitis or temporary blocking of the ureter may give the same kind of attacks.

For objective symptoms we have the urine alone to reply upon, in very many instances, but its change from normal, its admixture with blood or pus or bacteria or whatever it may be, tell us but part of the story. It does not tell us from which kidney or ureter, or whether from the bladder or urethra, the abnormal elements arise. Here, then, is indication for the cystoscope and urethral catheter, with which positive information is gained in the following manner:

It is a good rule first to employ the simple cystoscope, which is smaller of calibre and gives a broader field of observation. A general survey of the bladder is made and vesical complications, if present, are noted. The attention is then centered about the ureters, and indications of disease higher up are here carefully looked for. The mucous membrane around one or the other ureter may, for example, show ulceration or small tubercles, suggestive of descending tubercular infection of the kidney and ureter of that side. The ureteral lips may be thickened, and the orifice patent and devoid of its normal function of opening and closing as the little jet of urine is emitted, which gives evidence of some hindrance to the passage of urine below this point, and the possibility of consequent upward infection. The lips of the ureter may be pouting or its mucous membrane more or less prolapsed, which condition occurs with blocking of the ureter, usually by calculus not far above its orifice. Finally, we note with especial care the tiny stream of urine itself, the action of the ureter-mouth, the amount of urine, the intervals between the issued jets or drops, and the character of the secretion as it appears, whether bloody or mixed with pus, or thickened as by mucus. The difference between the two sides is then observed. If one or both sides fail to functionate such fact is noted.

In this way, before attempting to collect by ureteral catheterization the separate urines, we have come into possession of much valuable information, for we have ascertained that the trouble comes not from urethra or bladder, but from above these points. We have also gained evidence as to the side of the lesion and suggestions as to its nature. The ureteral catheter now gives us the means to determine more exactly the site of the lesion, and by analysis of the urine thus obtained, its nature. Without question the only accurate method of obtaining the separate urines without fear of admixture or contamination with the urine from the opposite side or from the bladder is by ureteral catheterization. This procedure is applicable and possible in the majority of cases. That it is accomplished with but little discomfort to the patient and little danger of infection stands to-day proved by ample experience.

Among those conditions of the kidneys brought within the scope of this means of diagnosis are: Renal tuberculosis, unilateral nephritis, pyelitis, pyonephrosis, pyelonephritis, hydronephrosis, atrophic kidney, single kidney, and at times new growths. In addition must be mentioned those instances where the ureteral catheter is used as a sound to detect abnormal constrictions and blocking of the ureter by strictures, stones, new growths, etc. Kelly, of Baltimore, says that he has demonstrated the presence of stone by bringing a ureteral catheter tipped with impressionable wax in contact with it. Tilden Brown and others have introduced the catheter incasing a fine metal stylet as far as the kidney pelvis, and then by a radiograph have demonstrated a dislocation of the kidney or cleared up the differential diagnosis between abdominal tumor and kidney.

Before speaking of the determination of renal sufficiency, the names of v. Koranyi, of Budapest; Casper and Richter, of Berlin; Kummell, of Hamburg, and Albarron, of Paris, should be mentioned as those men who by patient work and painstaking experiments have done the most in perfecting those means we now have at hand to estimate the functional ability of the respective kidneys. No more than a brief sketch of this subject will be attempted, as the writer's object is simply to bring the main points to the attention of those whose interest will lead them further in this line of thought and work.

We have seen how by ureteral catheterization we can make anatomical diagnoses of kidney affections; so is it possible to turn this same means to account in making functional diagnoses of the kidneys, which, in other words, means a measure of the amount of work of these organs—not of their united power however, but the estimate of what each individual kidney can do. This is the important thing from a surgical standpoint.

We are confronted in every case of surgical kidney disease by these questions: How much kidney parenchyma capable of work still exists? Is it sufficient to perform its function of ridding the body of the waste products of metabolism? How is this work divided and to what extent does each kidney participate? The importance of gaining this knowledge in every case of suspected kidney disorder of a surgical nature is emphasized when one sees in the literature what a number of cases have been reported, where only a single kidney is found, where the kidney of one side has in part or entirely degenerated, or where although the second kidney was present, it was so far diseased that on removal of the opposite organ it was incapable of performing sufficient work to maintain life. When to the cases which have been observed and reported are added those which have not come to autopsy, and therefore could not be reported, although they undoubtedly occurred, we see that it is possible very considerably to lessen the mortality in surgical kidney disease, if we are able to determine this question of sufficient or insufficient renal function.

The methods to this end which have been used and proved of practical value will be mentioned, and those means which are not to-day found useful, though worthy of our respect for having paved the way for better things, will be omitted.

The determination of the freezing point of the blood and urine (cryoscopy).—This method depends upon that physiological power possessed by the kidneys, which so regulates the osmotic pressure that it maintains in those fluids with which it has to do, namely the blood and urine, a constant measurable degree of molecular concentration which is determined by their freezing points. As the concentration of the fluid is increased, so as is its freezing point lowered below that of distilled water. It is to be understood when molecular concentration is mention that the concentration in an osmotic sense is meant, for a purely physical molecular concentration can exist without influence upon the point at which the fluid freezes, for example, the amount of albumen in the urine of kidney patients influences the specific gravity while it has no bearing on the lowering of the freezing point. A. v. Koranyi and others have shown that the blood of individuals with normal or sufficient kidney function freezes at — 0.56 degrees C., that the urine in these cases freezes at from — 1.0 degrees to — 2.0 degres C. Where the kidney function is insufficient, the molecular concentration of the blood is increased and the freezing point is lowered, and with this goes a higher freezing point for the urine.

When the normal freezing point of the blood tells us that there is enough healthy kidney parenchyma to free the body of its waste products, it does not show which kidney, or whether each kidney in part, is carrying on the work. For this information we rely on the freezing of the separate urines, along with the chemical and microscopical examination, the phloridzin test, etc.

Backmann's cryoscopic apparatus is the one ordinarily used. It consists of a cylindrical jar about 6 inches high by 4 inches or 5 inches in diameter. This is filled with a freezing mixture of ice and salt, in the centre of which is placed a tube about 1½ inches in diameter. In this latter is placed a somewhat smaller tube so that an air space between the two exists, insuring an even conduction of cold to the inner tube which contains the blood or urine to be tested. Fifteen to twenty cubic centimeters is the amount found most convenient, and in this is placed the thermometer with scale divided into hundredths of degrees; alongside of the thermometer is a mixer made of platinum wire and bent at its lower extremity so as to encircle the lower end of the thermometer; a cork perforated for the thermometer and mixer closes the tube.

The freezing point of distilled water is first determined and afterward that of the urine; the difference between the two is the freezing point sought. This for the urine is designated after the plan of Koranyi by Δ and for the blood by δ. While freezing, the specimen is constantly stirred by the mixer and the column of mercury is closely watched. It moves first rather slowly, but with increasing velocity it sinks until it has reached a point some distance below the freezing point, when in consequence of the warmth set free by freezing it rises to the point at which it freezes and there remains for a short time, during which the reading is made.

The usefulness of this method, as pointed out by Casper and Richter, is that it is a test of the renal activity as a whole, whereas the methylene blue or other similar tests show only the power of the kidney in one phase, and therefore give no idea of its natural functionating ability in eliminating the total of the solid molecules.

As mentioned before, it is the combination of ureteral catheterization and the freezing point determination of the separate urines which is of especial value in forming judgment as to the power of each organ; for it is the comparative values which we wish to obtain. The phloridzin test is an added proof of what each kidney is capable of, when used in conjunction with the freezing point determination. The action of this drug is chiefly on the kidneys and its result a so-called phloridzin-glycosuria. Small amounts (.005 grams), injected subcutaneously, are best suited for the results and are in no way harmful. The effect lasts but about three hours, beginning fifteen to thirty minutes after injection. The test is based on the amount of sugar excreted, but a delay in the sugar elimination is also a factor. The phloridzin method is useful because expressible in figures and comparative results of the two sides indicate the amount of functionating parenchyma present.

The elimination of nitrogen or its chief representative urea, when taken as a measure of the work of both kidneys, is of unstable value, for patients with kidney disease have periods of good and bad N-elimination. Even healthy individuals may have periods of retained nitrogen. On the other hand, an estimation of the amount of nitrogen from each kidney gives, in a diffential way, a valuable index of the renal ability, and considered with those methods already mentioned, forms an important link in that chain of evidence we seek.

SECOND GYNECOLOGICAL DIVISION.

Wm. M. Polk, *Gynecologist.*
Chas. C. Barrows, *Asst. Gynecologist.*
Geo. D. Hamlen, *Adjunct Asst. Gynecologist.*

In offering this report of the year's work on the Second Gynecological Division, it seems proper to say that the cases that have been subjected to operation are such as have been admitted to the hospital in its daily routine, or have been referred to the Division by members of the Attending Staff. No selection of cases has been made, and, as will be seen from the report, several patients have been received in a moribund condition. No effort has been made for records, and it is not the desire of the surgeon in charge of the Division to do more than to offer the cases, as they present themselves, for the consideration of those to whom they may be of interest.

It will be seen that 473 operations have been performed, the peritoneal cavity having been invaded in ninety-six (96) of these cases.

Twelve deaths have to be recorded, but it is only fair to state that of these four have occurred in post-partum septic cases, practically moribund on admission. Another death was the result of a Cæsarian section, transferred for operation from the Emergency Hospital, and operated on by one of the Emergency Hospital Staff. So that the mortality rate in percentage is hardly a fair test of the results of the work done on the Division, small as it really seems.

It will be seen that out of the sixty-six operations dependent upon the effects of inflammation of the tubes and ovaries, it was found necessary in seven cases to remove the appendix vermiformis. This ratio of, say, one case in ten, does not seem unusual. When one considers the very close proximity of the appendix and uterine appendages of the right side, it is not surprising to find that the inflammation of the uterine adnexa has involved in its course the appendix vermiformis, and so crippled that structure as to make its removal imperative, in a large proportion of cases coming into the hands of the gynecological surgeon.

In all of these cases the median incision was employed, the appendix being readily removed by this approach.

A fair inference may be drawn as to the opinion of the surgeon in charge of this Division, and that of his assistants, in regard to the treatment of posterior displacements of the uterus, by a study of the operative procedures which have been employed for the relief of this condition.

It will be seen that twenty-five cases have been subjected to some operative procedure looking to the relief of posterior uterine displacement. Of this number, in a majority of cases (viz., thirteen) it has been deemed best to resort to shortening of the round ligaments outside of the peritoneal cavity by means of Alexander's operation.

In ten cases the round ligaments have been shortened within the abdominal cavity. And in only two instances has it been found advisable to resort to other means of support.

In one case, occurring in a woman beyond the menopause, ventral fixation was resorted to, and in a second case the so-called ventral suspension was adopted.

It is the custom of this service to resort to other measures than that of establishing artificial adhesions between the abdominal parieties and the uterus for the support or suspension of that organ.

Seven cases of prolapse of the ovary (Fig. 1) have been treated by the method of operation suggested about two years ago by Dr. Barrows, the Assistant Gynecologist of the Division. This procedure is still under trial, but the results in the cases in which it has been adopted have been so satisfactory, both as to the relief of the distressing symptoms dependent on ovarian prolapse, as well as to the subsequent life of the patient, general and menstrual, that it seems justifiable to offer in detail a description of the procedure.

The abdomen is opened under the usual antiseptic precautions, an incision of two inches being usually sufficient. The patient is then placed in the Trendelenberg posture and the intestines held well out of the pelvis by a wall composed of three bolsters of gauze, as seen in Figure 3. These bolsters, when properly applied, hold back the intestines so as to admit of easy and comfortable work within the pelvic cavity. The round ligaments are then caught up and shortened, as seen in Figure 2. The next step of the procedure consists in shortening the infundibulo-pelvic

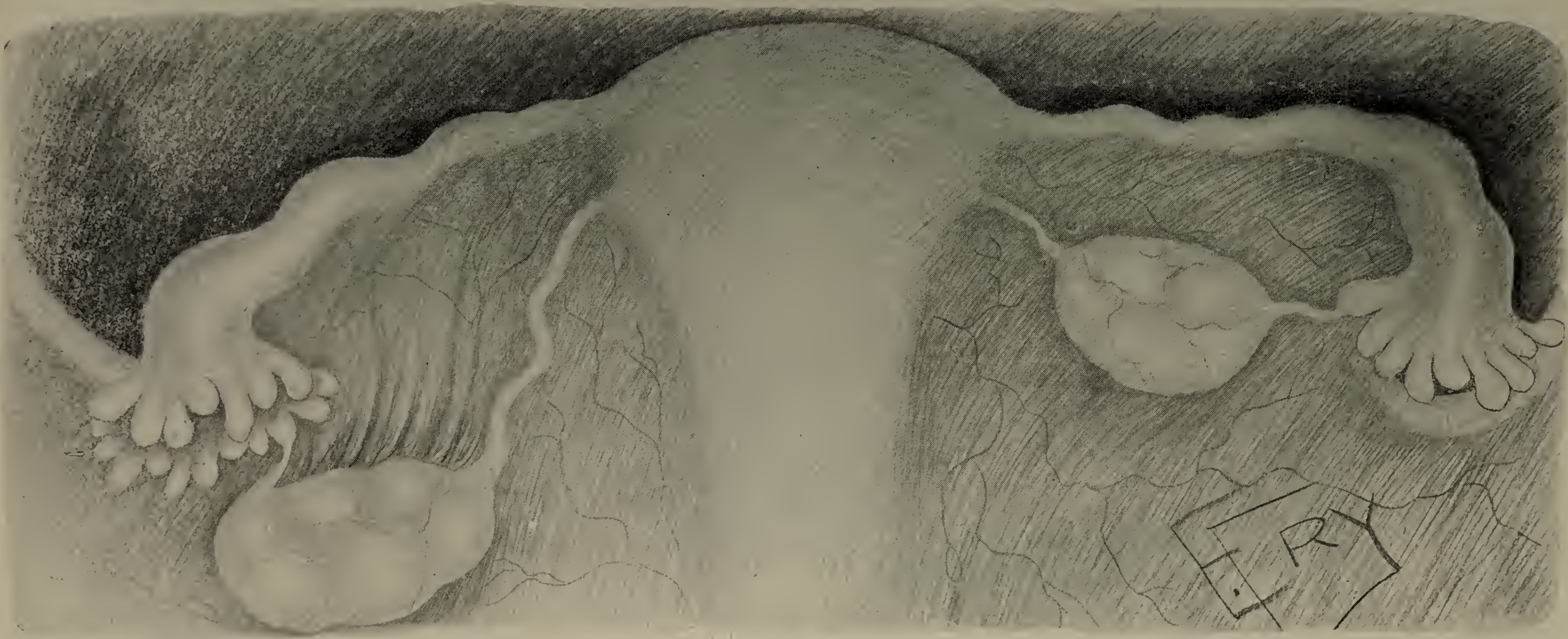

Fig. 1.

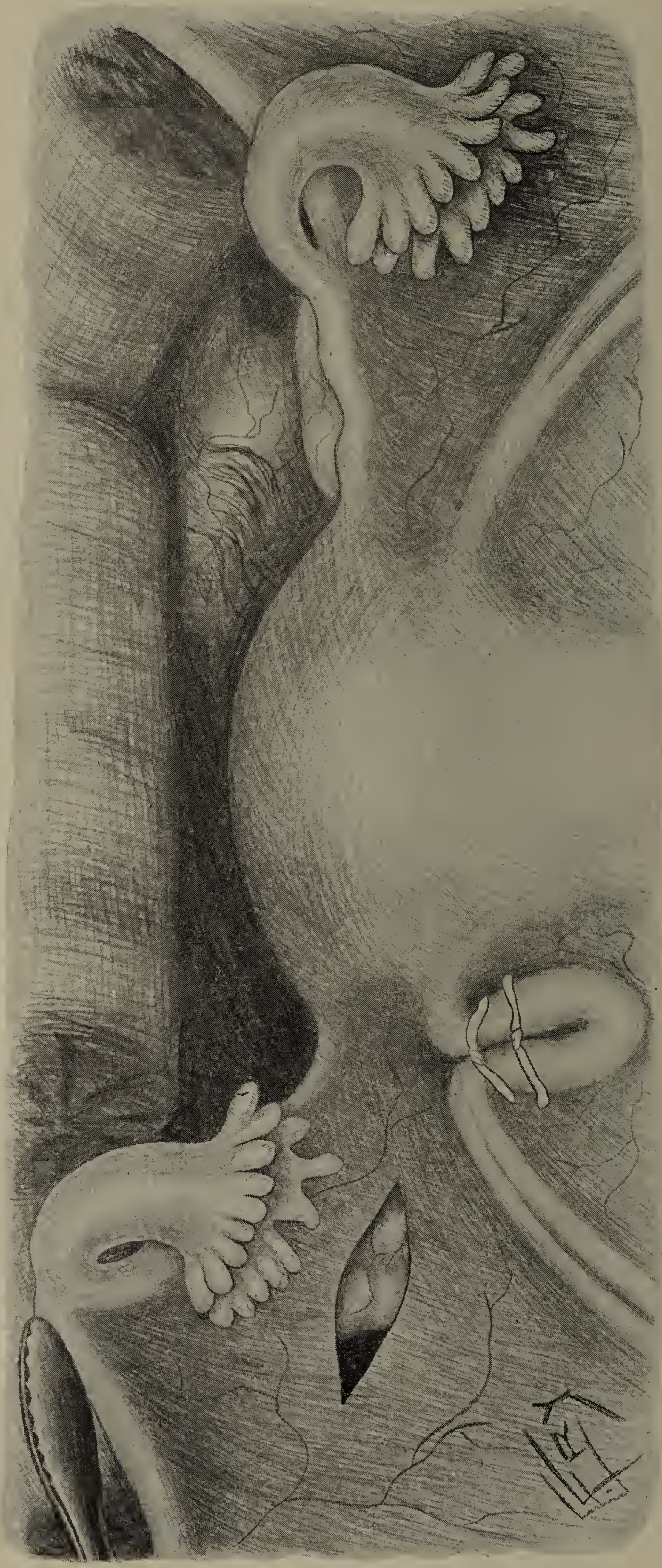

Fig. 2.

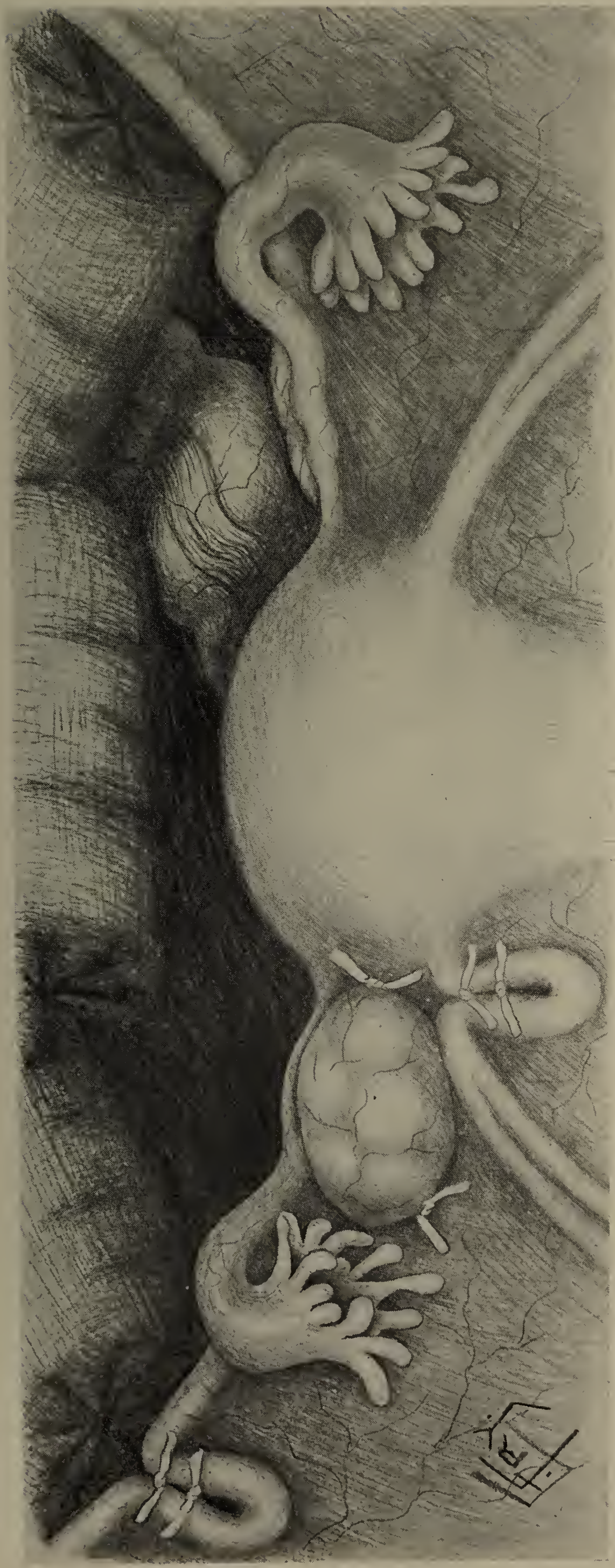

Fig. 3.

ligament, after the same manner as that followed in shortening the round ligaments, as shown in Figure 3.

In this way, it will be seen that a V-shaped double fold of peritoneum is stretched between the round ligament and the infundibulo-pelvic ligament and immediately above the prolapsed ovary lying below it in Douglas' pouch. An incision is then made through this double fold of peritoneum, say, an inch long, and the ovary is brought through it and placed upon the shelf thus provided for it. A single catgut or small silk suture at each end of the incision serves to limit it and at the same time steady the ovary in its new position. In one case pregnancy and successful delivery has occurred in a patient, the subject of a double operation of this kind; and in all of the cases the distressing symptoms dependent on ovarian prolapse have been promptly and apparently permanently relieved.

This procedure was originally suggested for the surgical treatment of simple non-adherent prolapse of the ovary, but it may be readily seen how serviceable it may be in cases of prolapsed adherent ovaries, where the breaking up of adhesions is almost certain to be followed by the formation of new adhesions between surrounding structures and the denuded surfaces of the ovary, but recently freed from the adhesions about it. This procedure places the ovary safely on the top shelf of the pelvis, where it may carry on its functions without interruption or interference.

The case of Cæsarian section, which was transferred from the Emergency Hospital to Ward 23 for operation, presents a condition as rare as it is interesting.

The patient, an Italian woman, of thirty, a primipara, was brought to the Emergency Hospital by ambulance at 1 o'clock in the morning of June 12, 1904. The patient had been in labor fourteen hours prior to her admission, in the hands of a midwife, and subsequently under the care of two physicians of her own race, who had made repeated unsuccessful attempts at instrumental delivery. After admission to the Emergency unsuccessful attempts at instrumental delivery were made by the house physician and by Dr. Moore, of the visiting staff. The patient was believed to be a proper subject for the Cæsarian section, and was transferred to Bellevue Hospital for operation. On admission to Ward 23, at 8.30 A. M., the patient's pulse was 134, temperature 101.02, and respiration 44. At 7.10 A. M. the patient had had a severe chill, followed by great physical depression. Following this the fœtal heart, which had been easily counted prior to this time, became unappreciable. The patient was seen by Dr. Barrows shortly after this, and in spite of the fact that the fœtal heart could not be satisfactorily heard, Cæsarian section was deemed advisable. This opinion was strengthened by the fact that it seemed possible that the patient might be the subject of an incomplete rupture of the uterus, demanding operative interference. The courtesy of the Division was extended to Dr. Moore, who performed the operation.

On opening the abdomen the uterus was found intact. The uterus was delivered through the incision and opened by the usual median incision. The placenta and membranes were quickly shelled out intact without additional rupture, as suggested by Dr. Barrows in a report of a Cæsarian section in 1901, the wound being thus kept clean and free from infection from the intramembranal contents. Where the membranes have not been ruptured this is, of course, absolute; but in cases such as this, where there has been already a rupture of the membranes, the separation must be followed carefully into the cervical portion of the uterus, and measures taken to prevent the escape of the contents of the sac at this point. On opening the sac the child—a well-formed male of 8 pounds—was found to be dead. Dr. Hamlen, to whom it was intrusted, called the attention of those present at once to the fact that *rigor mortis* existed in a marked degree. The upper and lower extremities, which were flexed upon the trunk, were so rigid as to make it practically impossible to extend them, and the child was in complete *rigor mortis.*

This condition of *rigor mortis* in the fœtus is of such rare occurrence as to make a report of this case desirable.

Edgar, in his "Practice of Obstetrics," quotes Ballantyne as having seen one case and reported twenty-five others, several occurring in connection with Cæsarian section.

It may be readily understood how so marked a fœtal rigidity as existed in this case might easily be a source of serious fœtal dystocia. That this *rigor mortis* in the fœtus may be of rapid development seems to be proven by the fact that the fœtal heart could be readily appreciated by the members of the staff present, less than two hours prior to the operation.

This patient did perfectly well until eighteen hours after the operation, when, after declining to take a stimulant offered her, she suddenly expired.

Unfortunately, an autopsy could not be obtained.

	July.	Aug.	Sept.	Oct.	Nov	Dec.	Jan.	Feb.	Mar.	Apr.	May.	June.	Total.
Alexander's operation	1	..	..	1	2	..	2	2	3	1	1	..	13
Amputation of breast......	..	1	..	..	..	1	..	1	..	1	..	1	5
Amputation of cervix.............	..	1	1	..	2	..	..	1	2	1	1	..	9
Amputation of labia minora.......	..	..	..	..	..	..	1	..	..	..	..	..	1
Appendectomy.......	1	1	1	1	5	..	4	1	2	3	..	..	19
Barrows's operation for prolapsed ovaries	..	..	1	2	1	2	1	..	..	..	..	..	7
Braxton-Hick's operation for central placenta praevia ...	..	..	..	..	..	1	..	..	..	..	..	..	1
Breaking-up of pelvic adhesions...	2	1	1	..	..	2	..	..	..	1	..	..	7
Cholecystectomy	1	..	..	..	..	..	..	..	..	..	..	..	1
Cholecystotomy..................	1	..	..	..	..	..	..	..	..	..	..	..	1
Caesarian section.....	..	..	..	..	..	..	..	..	..	..	..	1	1
Circumcision	..	..	..	..	..	..	..	..	..	1	..	..	1
Closure of fistula-cervico-vaginal.	..	..	..	..	..	..	1	..	..	..	..	..	1
" recto-vaginal...	1	1	..	..	..	..	..	..	..	..	..	..	2
Colporrhaphy, anterior...........	..	..	..	..	1	..	2	1	..	..	1	..	5
" posterior.........	..	1	1	..	1	..	..	..	..	..	..	..	3
Colpotomy, lateral................	..	..	..	..	..	..	..	1	1	..	..	..	2
" posterior.............	2	3	..	2	3	2	3	3	3	1	1	..	23
Curettage........................	18	10	10	1	11	7	12	11	18	14	15	5	145
Curetting of old abdominal sinus..	..	..	..	..	..	..	..	2	1	..	..	..	3
Cystoscopy	1	..	..	..	.	..	..	..	..	..	..	..	1
Dilation of cervix, curettage and glass plug..................	..	..	2	..	2	..	2	2	1	2	3	1	15
Dudley's operation	..	..	..	..	1	..	..	..	..	1	..	..	2
Excision of adenoma, breast......	..	..	..	..	..	..	..	..	..	..	1	..	1
Excision of varicose veins of legs.	..	..	..	..	..	..	..	..	..	1	..	..	1
Excision of vulvo-vaginal abscess.	..	1	1	1	..	..	..	..	..	1	..	..	4
Excision of cervical glands, tubercular	..	..	..	..	..	..	..	1	..	..	..	..	1
Excision of vulvo-vaginal cyst ..	..	..	..	1	..	..	..	..	..	1	..	..	2
Hernia, radical operation for, inguinal....................	..	..	..	1	1	..	..	1	..	..	1	..	4
Hernia, radical operation for, ventral...............	..	1	..	1	..	..	..	..	..	1	..	..	3
Hysterectomy, abdominal	1	..	..	3	..	3	2	..	..	..	..	1	10
" vaginal...........	..	1	..	1	..	..	..	..	..	..	..	..	2
Hysterorrhaphy..	..	1	..	..	..	..	..	..	..	..	..	..	1
Incision and drainage of abscess, appendicular................	..	..	..	..	1	..	..	..	..	1	..	..	2
Incision and drainage of abscess, (a) linguinal, (b) ischio-rectal	..	..	..	(a)1	..	..	..	..	(b)1	..	..	..	2
Incision and drainage of abscess, (a) vulvo-vaginal, (b) periurethral....................	1(a)	..	..	(a)1	..	..	..	..	..	(b)2	..	..	4
Incision and drainage of retroperitoneal hematoma......	..	1	..	..	..	..	..	..	..	..	..	..	1

	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May.	June.	Total.
Laparotomy, exploratory..........	..	1	..	..	2	..	1	..	..	..	1	..	5
Lumbar incision exploratory.....	..	..	..	..	..	..	..	..	..	..	1	..	1
Nephrectomy.....	..	.	1	..	..	..	..	..	..	2	..	..	3
Nephropexy..	..	..	..	1	..	..	1	..	..	..	1	..	3
Perineorrhaphy	2	4	4	3	3	1	4	5	3	3	2	..	34
Protoscopy.	1	..	..	..	..	..	..	..	..	..	..	..	1
Rapid emptying of uterus, placenta praevia...............	..	..	..	..	..	..	2	..	..	..	..	..	2
Rapid emptying of uterus, inevitable abortion...........	..	..	..	..	..	..	..	1		..	..	..	1
Removal of cervical carcinoma by cautery.	1	..	..	..	..	..	..	..	..	..	..	..	1
Removal of cyst, intraligamentous	..	..	..	..	1	..	..	..	..	..	..	..	1
" ovarian, large...	..	..	..	..	..	..	..	1	..	1	..	1	3
Removal of cyst, hematocele of broad lig., and repair of ligament.......	1	..	..	.	..	..	..	..	..	..	..	..	1
Removal of hemorrhoids, by clamp and cautery....	1	..	..	2	1	..	..	..	..	..	..	..	4
Removal of hemorrhoids by igni-puncture	..	1	..	..	..	..	..	..	..	..	..	..	1
Removal of hemorrhoids, by ligation and excision..	2	..	..	..	..	..	1	1	..	1	..	1	6
Resection of ovaries....	1	2	1	2	1	1	..	1	1	..	..	..	10
Salpingectomy, single...........	1	3	..	2	1	..	1	1	2	1	1	..	13
" double	..	..	..	1	..	..	..	..	..	..	..	..	1
Salpingo-oöphorectomy, single....	5	1	..	4	5	2	3	2	8	2	1	1	34
" double...	1	..	2	1	4	3	1	..	2	2	..	2	18
Tonsilectomy	..	..	..	..	..	..	..	..	..	..	..	1	1
Trachelorrhaphy.....	3	1	..	1	..	1	3	1	..	1	1	..	12
Ventral suspension......	..	1	..	..	..	..	..	..	..	..	..	..	1
Wylie's operation, internal short. of round ligaments...	..	3	1	...	1	2	..	2	..	1	..	..	10
Total...........	49	41	27	47	50	28	47	42	48	47	32	15	473

RENAL TUBERCULOSIS.

Service of Dr. F. Tilden Brown.

C. C.; age, 39 years, Nova Scotia; sea captain. Admitted January 9, 1904. Discharged, improved, March 2, 1904.

Diagnosis—Left renal tuberculosis.

Family history—Negative.

Habits—Excellent.

Previous history—Remembers no other serious illness. No venereal disease. For five years has complained of pain and weakness in back; upper lumbar region. Finds it difficult to stand upright; relieved by stooping. For some years "rheumatic pains about heart" which spread about chest; attacks would last two days. No palpitation or shortness of breath. No pulmonary symptoms. No gastric symptoms.

Two years ago an attack, lasting a week, of pain starting in left testis running into abdomen and down left thigh; chill, fever, vomiting. Some frequency of micturition.

One year ago he had a chill and fever, with pain in left side. Was prostrated for a month. At this time there was a free discharge of pus in the urine and frequency of micturition. Some cold sweats at night. Entirely recovered. Never passed stone or gravel.

Present history—Six months ago began to pass blood in urine, coming most often at the end of urination. Also to have attacks of pain in left side of abdomen running down to left testis. Has had chills and fever at irregular intervals. Passes urine containing pus; at times, cold sweats at night.

Frequency of micturition most marked during day when moving about. Pain at the end of urination which extends from perineum to end of penis. Has lost some flesh and strength.

Physical examination—Negative.

Abdomen slightly distended; tenderness and slight resistance to deep palpation and an indefinite sense of mass in region of left kidney; some tenderness in suprapubic region; no mass.

January 12, urine shows albumen, 1 gram per litre; pus and tubercle bacilli.

Cytoscopic examination shows a hyperæmic and swollen irregular surface about the rather patulous left ureter mouth.

Synchronous ureter catheterization gave right ureter specimen: Clear urea, 1.4 per cent.; freezing point, – 1.69 degrees centigrade; no pus, no blood, no bacteria, no casts. Left ureter specimen: Cloudy urea, .7 per cent.; freezing point, – .94 degrees centigrade; pus, tubercle bacilli, no casts.

January 27, freezing point of blood, – .61 degrees centigrade.

Patient improved under hospital care and decided to return to his home without operation. During March and April he was at home in Nova Scotia under good hygienic and dietetic conditions, but symptoms became more marked again and he returned to New York, entered another hospital and requested operation.

May 15, freezing point of blood, – 55 degrees centigrade.

May 16, before operation, urine examination showed 50 ounces as the average amount per diem, 1020, acid, amber, slightly turbid, heavy white precipitate, heavy trace albumen, no sugar, pus, 2 per cent. No tubercle bacilli demonstrable. (Leucocytes, 10,000.)

Operation, May 18, Dr. Brown. Gas and ether. Nephrectomy.

Incision parallel with lower ribs. Kidney was greatly enlarged and lobulated. Each lobule seemed filled with semi-solid material, and much care was necessary to aboid rupture during removal.

Pedicle was with difficulty isolated and chromic gut ligature applied to whole pedicle. Ureter and renal vessels were then separately ligated. Condition good throughout.

May 26, temperature has not been above 100 degrees since second day. Very little discharge; wound in excellent condition; urine passed in good amounts (50 ounces). Very little pus; no tubercle bacilli demonstrable.

June 22, perfect primary union except at posterior extremity where now small superficial granulating area exists; this is rapidly healing. Feels well, stronger than for many months; micturition normal. Returns home to-morrow.

A DEMONSTRATION OF SYNCHRONOUS URETER-CATHETERIZATION IN A MALE.*

F. Tilden Brown, M. D.

When invited by the essayist of the evening to discuss his paper it was felt that the topic would be so exhaustively treated that our brief remarks might better be limited to some allied subject. Hence this explanation of the speaker's cystoscope and his technique in its employment for synchronous ureter-catheterization.

Gratifying evidence of the growing appreciation of this rather long neglected but most valuable branch of cystoscopy—ureter-catheterization—is to be seen in the recent action taken by the medical and governing boards of a few of our larger hospitals, whereby good equipments for such work have been installed in special quarters and the whole plant relegated to the sole management of a competent chief.

When the many other institutions follow this example of thus safe-guarding their patients, it is predicted that those occasional misfortunes, where a single kidney or the better of two has been removed, will not be heard of in the future. Even if such so-called accidents are gross and rare, they nevertheless occur. But such an occurrence is only a crude example of the many lesser errors which the diagnostic possibilities of the topic under discussion are competent to forestall. Moreover, many refinements in renal diagnosis, both organic and functional, are made possible only by this means. Nor can its prognostic services be overlooked where the adequacy or inadequacy of the other kidney becomes a most important factor in some meditated renal operation.

We would like, if time permitted, to review the several various physiologico-chemical tests applied to the separate urines for the determination of disease or insufficiency, but the immediate aim must be directed simply to showing our mechanical methods for securing these separate excretions.

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*Made before the Medical Association of the Greater City of New York at the Academy of Medicine January 11, 1904.

There are not a few who think that ureter-catheterization is much more in evidence as a topic than as a practical everyday reality. Believing that this assumption is mainly supported by the great number who have never seen it done, and who at the same time infer that marked discomfiture if not danger attends its accomplishment, we shall appreciate having the brief indulgence of this society while catheterizing the ureters of a male patient, and in so doing hope to show that the speaker's instruments, made in this city, possess a wider range of applicability than do those of European design.

The patient is here comfortably postured, with his legs flexed and supported by a crutch under each knee (Fig. 1). Various details of this particular table have been elaborated for me by the Kny-Scheerer Company. It possesses some features which make it a desirable table for infra- and supra-pelvic operations, as well as for examination of the same regions (Fig. 2).

When the patient's body and limbs are made to feel secure and at rest local anæsthesia of the lower urinary tract is all that is ever required, with very rare exceptions, for ureter-catheterization. Here we are using 3 per cent. eucaine solution.

By suspended bottle syphonage the bladder and urethra have just been irrigated with a warm boric acid solution; as this fluid now returns perfectly clear three ounces of it are left in the bladder, and the lamp-bearing sheath of the cystoscope, closed by an obturator, is taken in hand, after testing its cold lamp, the whole is lubricated and passed to the bladder. Now the obturator is withdrawn and the catheter telescope (previously prepared with two flexible 18-inch long catheters) is substituted for the former. During this intercharge of obturator for catheter-telescope you notice that the boric acid solution does not escape through the open sheath, because I raise the occular end, which brings the vesical open end in contact with the bladder floor, thus occluding it. When now the catheter-telescope is all but pushed home, I lower the occular end and so prevent any impact upon the vesical wall as the insertion is completed. It would make no difference, however, if the solution should escape from the bladder for, by connecting the hose to one of these irrigating nozzles on the side of the sheath, a fresh supply can quickly be

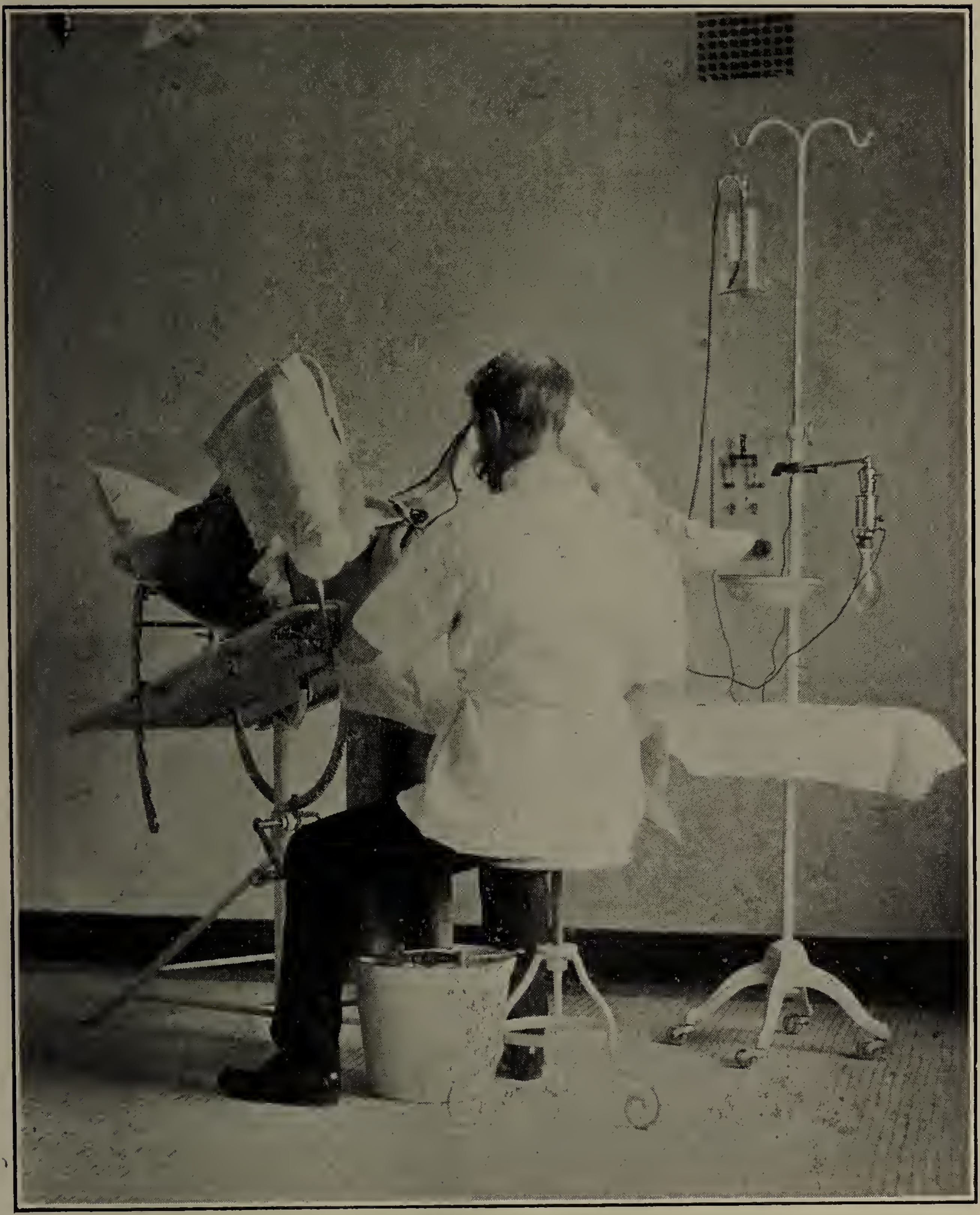


Fig. 1. Patient postured for ureter-catheterization on the writer's table. Electric cord and irrigating tube suspended over the patient's left thigh.

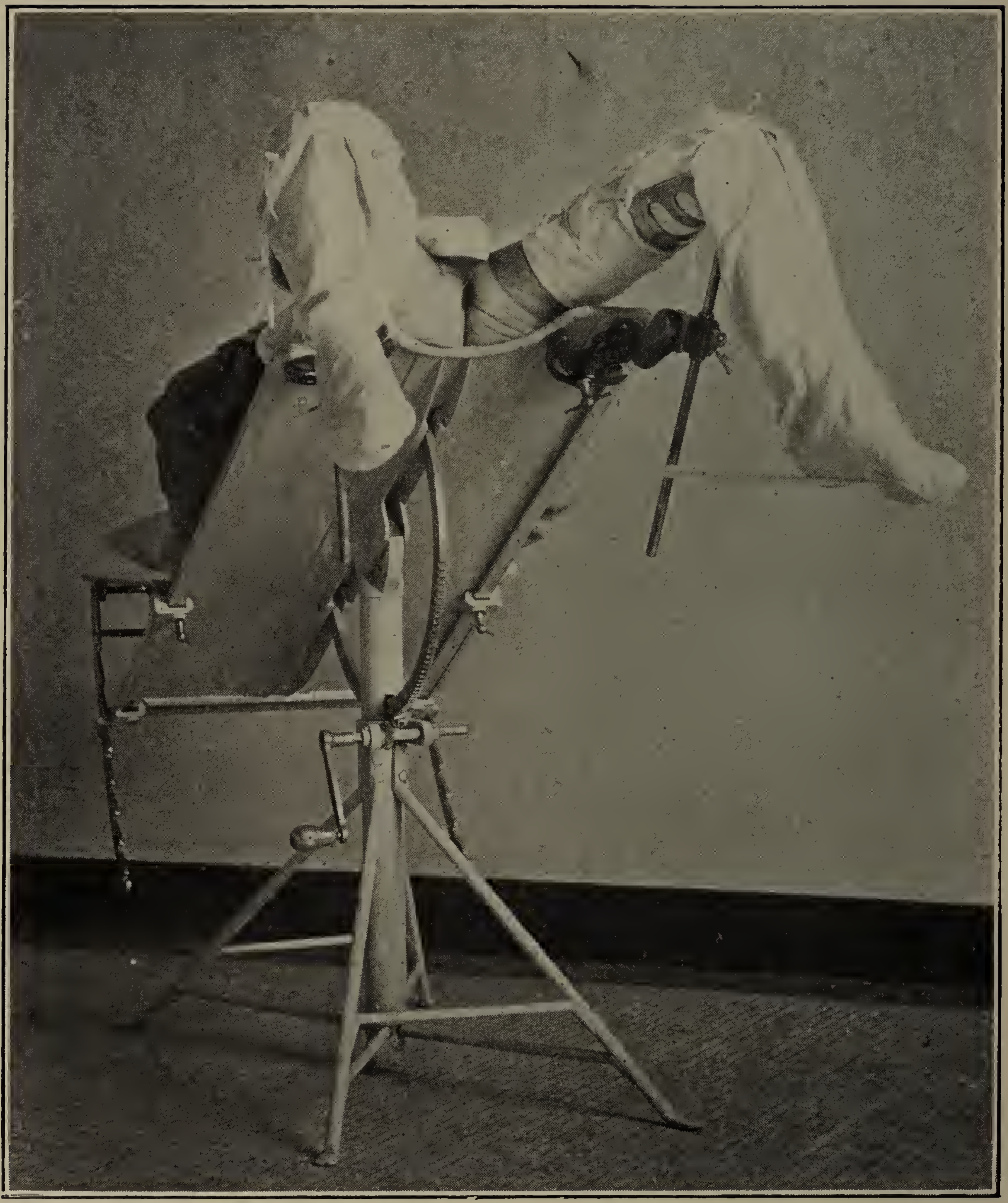


Fig. 2. Modified Trendelenburg's posture for supra and infra pelvic operation or examination.

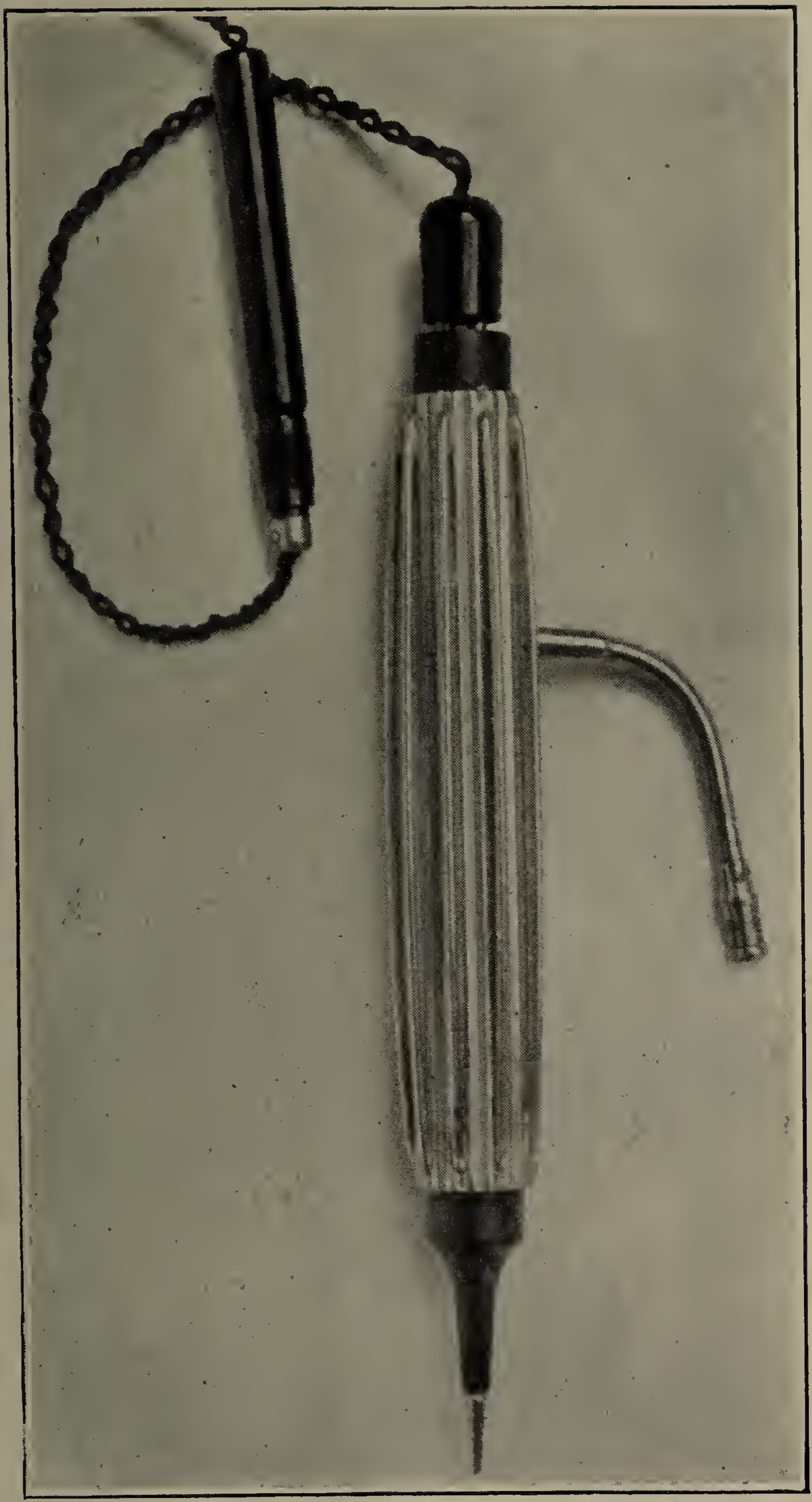


Fig. 3. The writer's Cylinder for Heating Air by Electricity. The metal plug point fits into either of the irrigating cocks on the sheath of the cystoscope, while the metal side tube connects with rubber tubing from the pump.

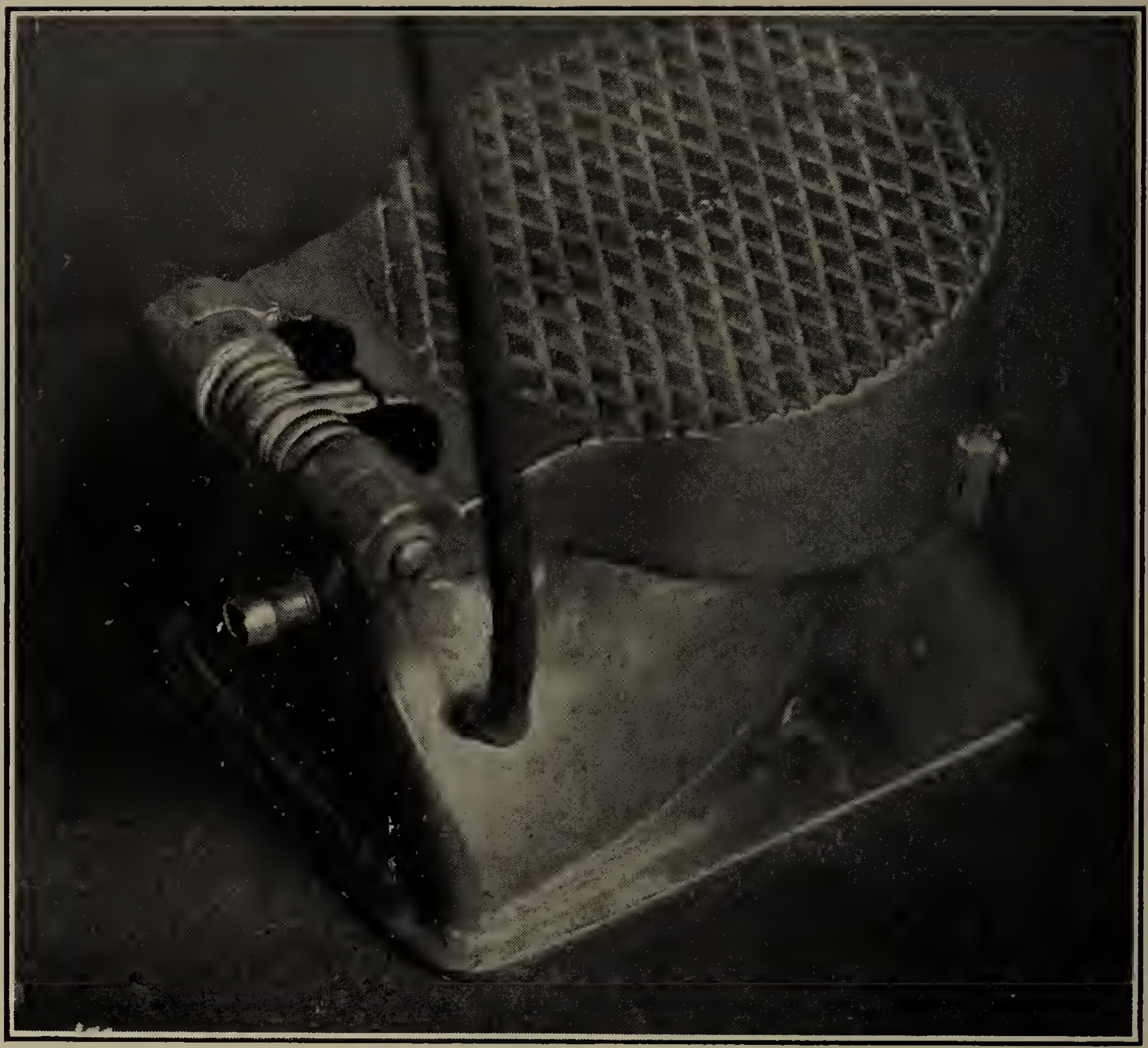


Fig. 4. Foot Air Pump. Connecting by tube with the heating cylinder.

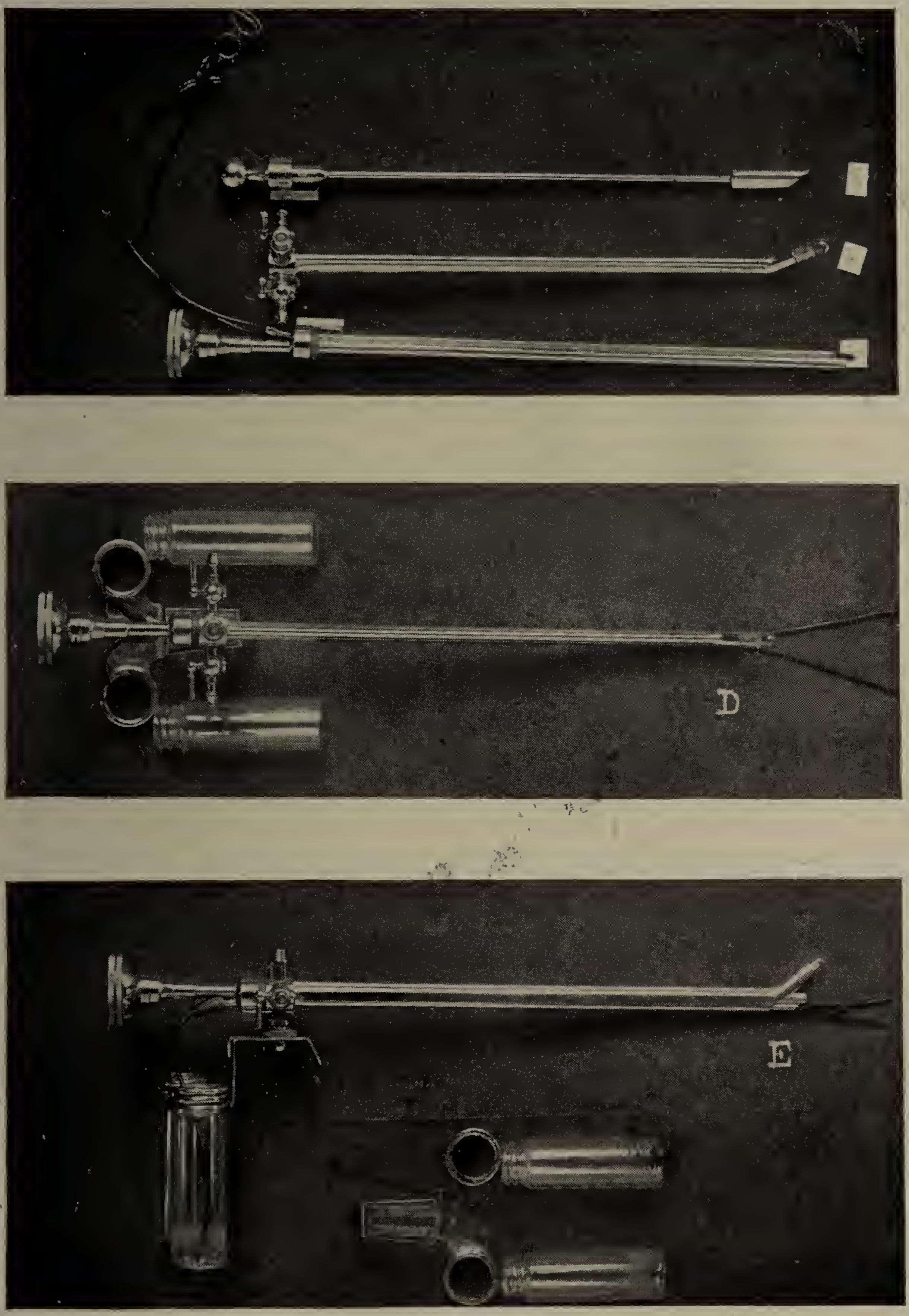
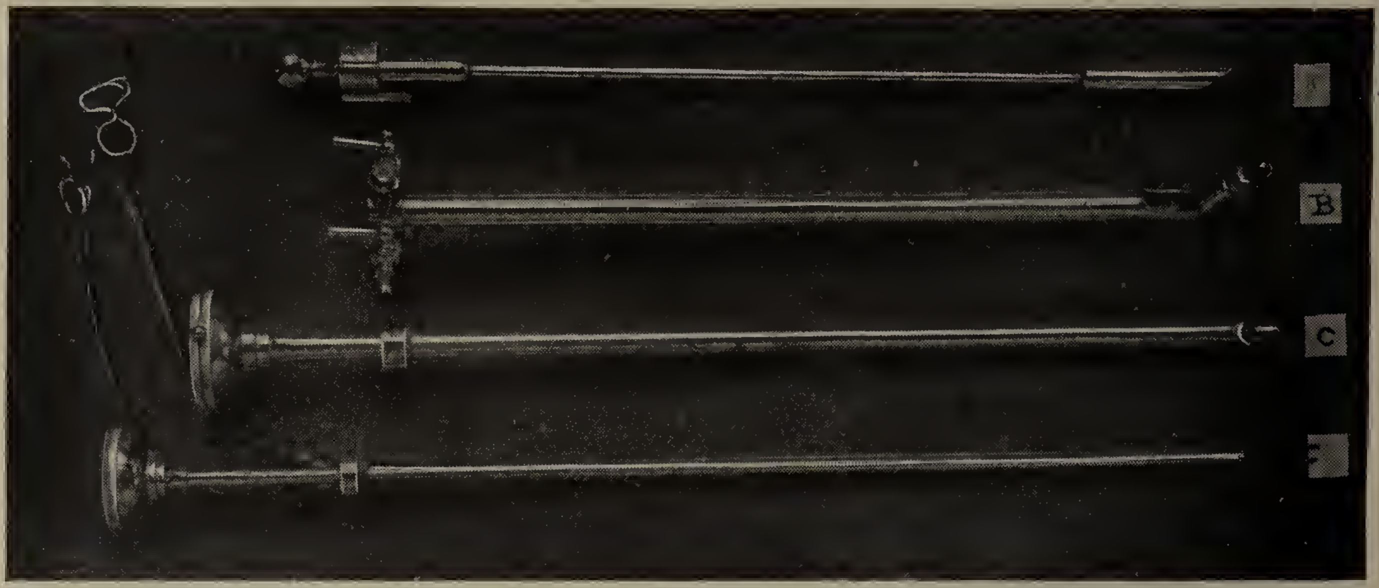
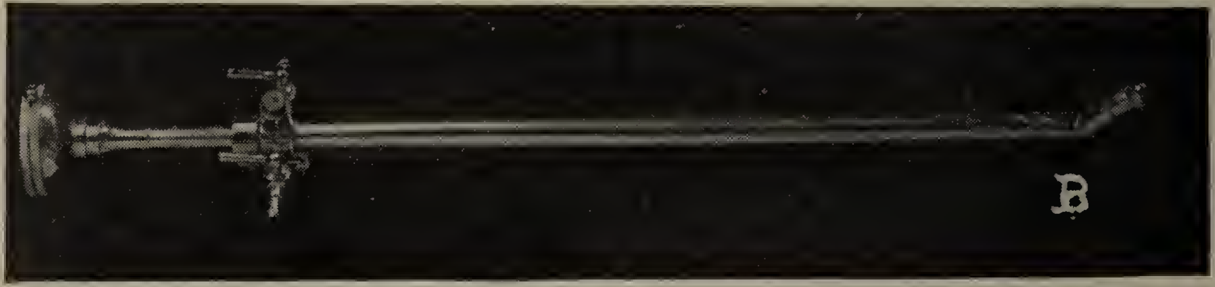


Fig. 5. The writer's Oval Sheath Catheterizing Cystoscope. Cut E shows in profile the projection of the telescope and catheter canals beyond the sheath for the urposes explained in the text.





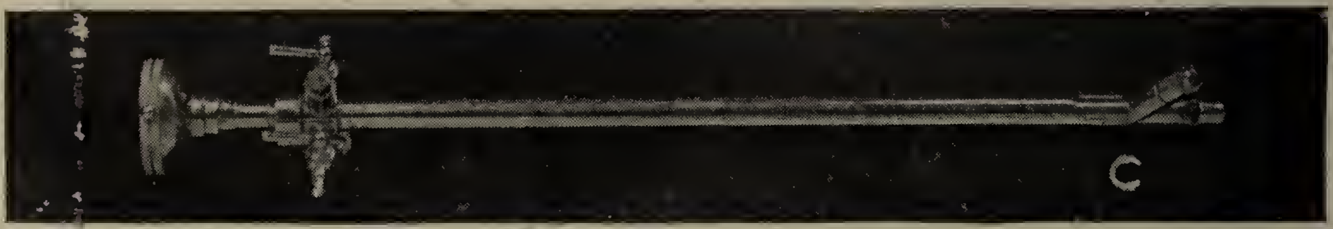


Fig. 6. The writer's Composite Cystoscope, Cut B, shows its Revolving Prismatic Telescope proximal to the open lamp. Cut C, the same Telescope pushed in and distal to the lamp.

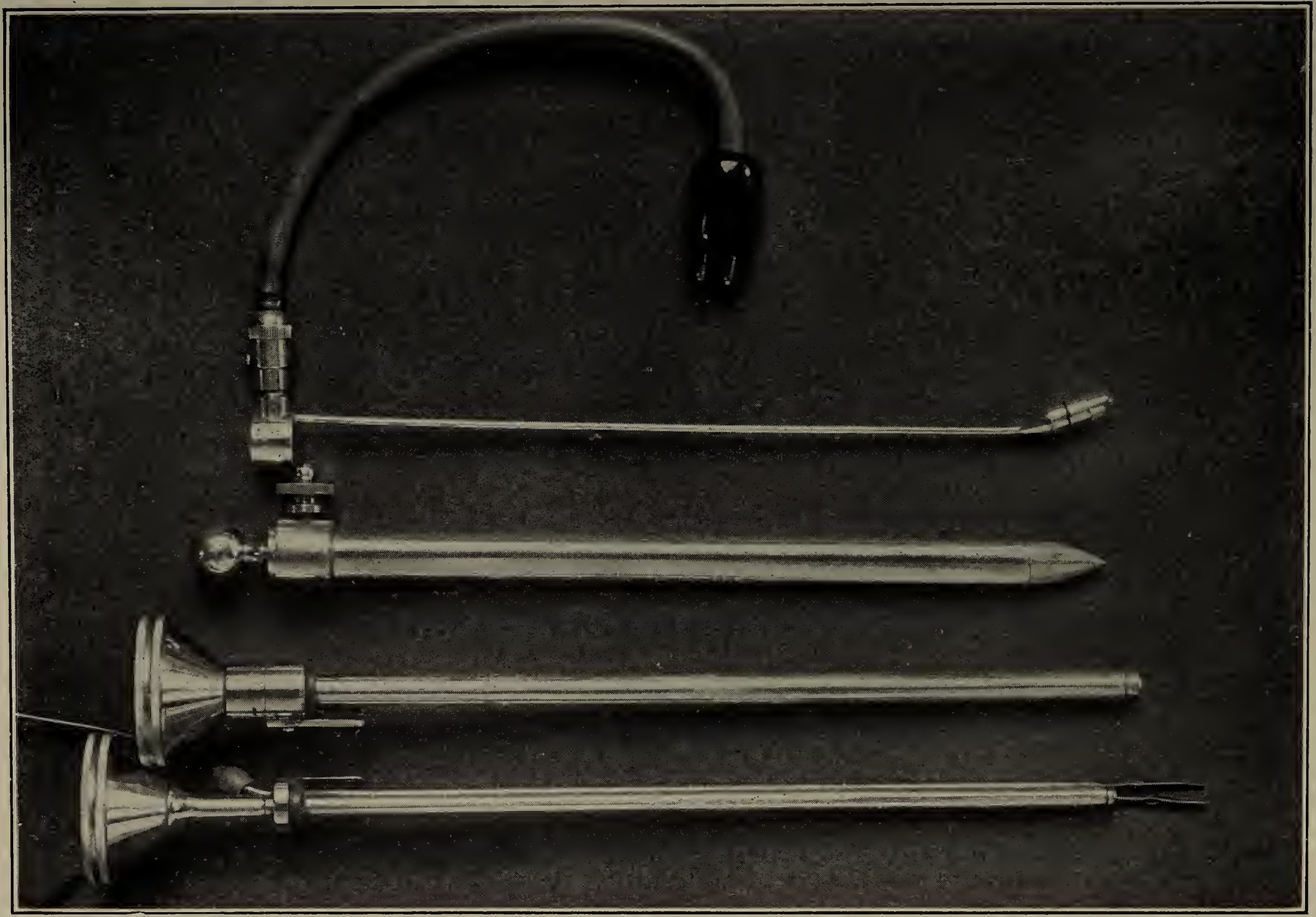


Fig 7. The writer's Supra Pubic Trocar and Canular Cystoscope.

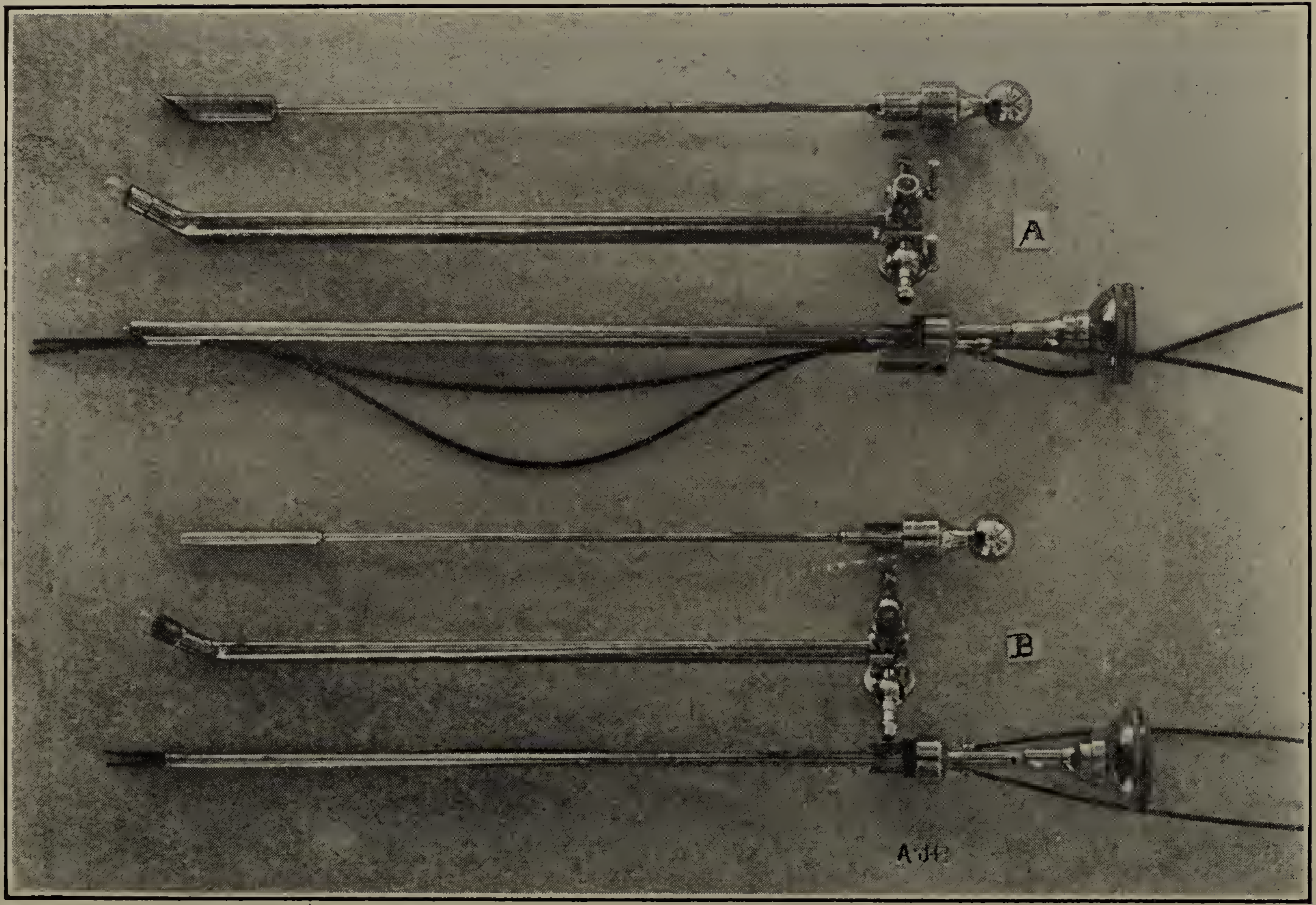


Fig. 8. Two circumferential sizes of the author's Oval Sheath Cystoscope:

A. Parts of the Twenty-four "French" size—viewed laterally. Catheters are drawn down to show the open part of their canals.

B. Parts of the Twenty "French" size—viewed more vertically. This small size is adapted for a child's or strictured adult's urethra.

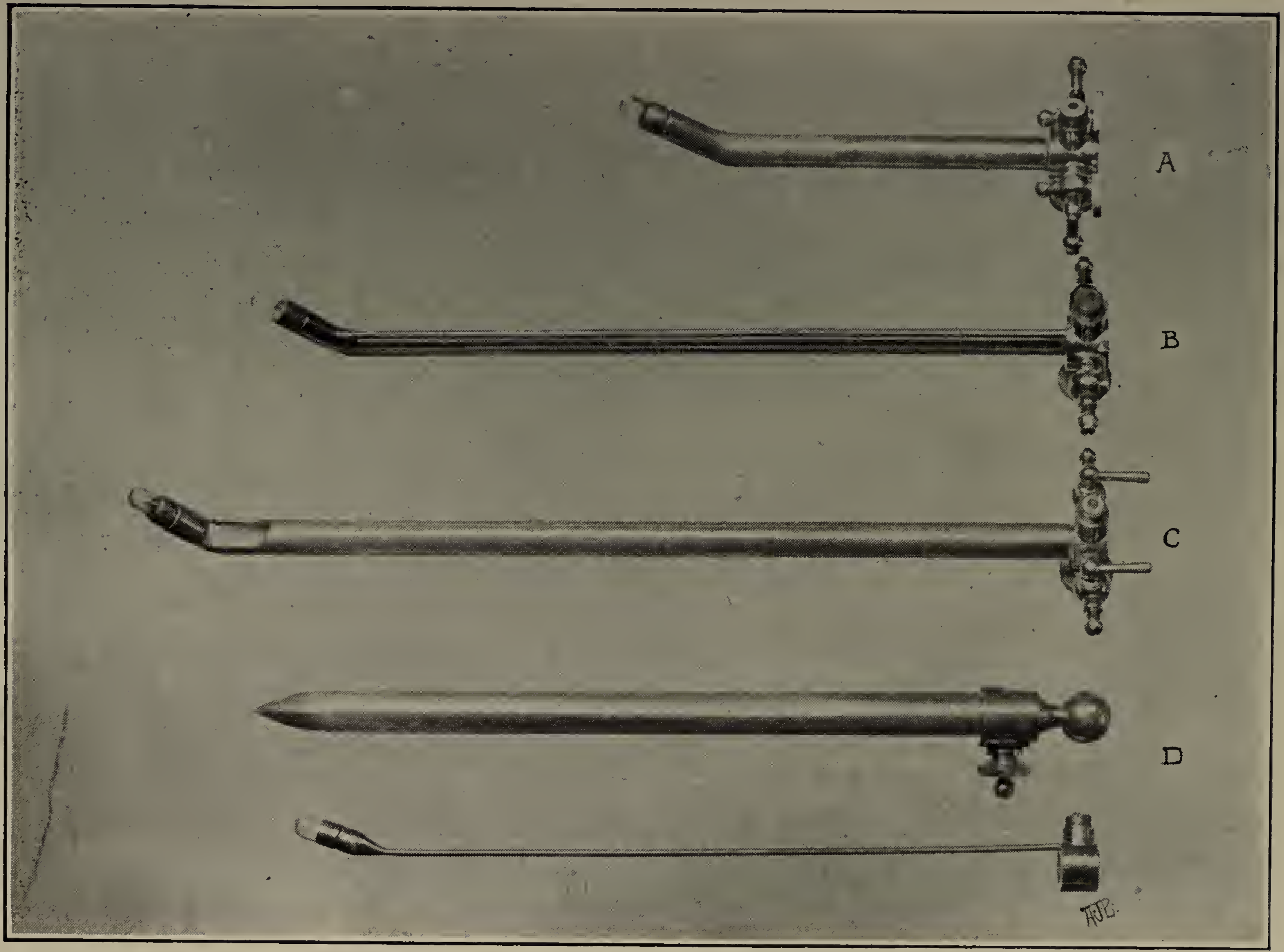


Fig. 9. Some extreme types of the author's Composite Catheterizing Cystoscope :

A. Short cylindrical sheath ; for female cases only. Twenty-four French.

B. Very small sheath (Twenty French). For use in children and the strictured adult urethra.

C. Extra long sheath ; fenestrated for a prismatic and retrograde telescope. Especially for prostatic cases.

D. Oval trocar and canula sheath with insertable lamp. Its telescopic parts shown in Fig. 7. For supra-pubic puncture cystoscopy.

replaced. The readiness with which an interchange of distending medium, water or air, can be effected with this type of instrument, whether the telescopic tube is in the sheath or not, is one of the several advantages it possesses over my first double catheterizing cystoscope. I may here call attention to another feature of this system, in that, when the catheter-telescope was just now introduced, there was no possibility of its catheter channels or of the tips of the catheters catching up infectious material in the urethra, because the sheath through which they were passed formed a continuous lining to the canal from meatus to the bladder. Now, that the necessary component parts of cystoscope are in the bladder, we support its occular end with the left hand and turn on the light with the right, taking at once a hasty view with the left eye of the fundus, so as to bring the lens to focus on the left horn of the trigonum, toward the middle of which we will identify the mouth of the corresponding ureter.

In bringing the intra vesical lens to this position, you notice that the ocular end of the cystoscope has gradually been moved pretty close to the patient's right thigh, and because of this proximity, there is not much room for one's head, consequently that eye—namely, the left—which is nearest to the patient's right leg is the one here used. At this moment, when the left ureter mouth is quite clearly seen, the forefinger and thumb of the right hand grasp the left catheter close to the cystoscope and push it forward, the catheter point first comes into view at the margin of the intravesical lens, advanced a bit more and it comes to the middle of the lens, and about the same time it is seen to engage in the ureter mouth; continued hence until two and a half inches have entered the ureter, when, without changing the vertical inclination of the cystoscope, its vesical lens is carried along the curved margin of the trigonum toward the right horn, until the ocular end of the instrument approaches the left leg, then the examiner changes to the use of his right eye, at the same time taking the instrument in the right hand, so as to have the left thumb and finger ready to advance the right catheter the moment the right osculum or the papilla indicating it comes into sight. By careful observation of the vesical landmarks during this excursion of the cystoscope the second ureter mouth is nearly always more quickly found than was the first. Now, having passed the right catheter to a depth of two inches, the cystoscope is brought back into the exact median line and there held by the left hand steadied against the patient's thigh or by the forearm on the table. This care is taken in order to minimize irritating movements of the instrument, as well as any tendency for it to go deeper into the bladder and so unduly bend the catheters or stretch the ureter mouths.

Now that both catheters are in place, I remove the rubber caps from their outer ends. Up to this moment the lumen of each catheter has been thus sealed to prevent any vesical fluid gaining access while their tips were exposed in the bladder.

Ordinarliy, two culture tubes are inoculated by four or five drops coming from each catheter, then the free ends of the catheters are turned into these 10 c. c. sterile bottles, held by a yoke attachment to the cystoscope (Fig. 5).

You may see here what is commonly the case in normal kidneys, that while five or six drops are coming rapidly from one kidney there is no output from the other; that is, the action of the two glands is quite regularly alternating.

During this collection of the urines a sensitive bladder can be given comfort by opening the stop-cock of one of the irrigating canals, to allow the distending medium to slowly escape. If, as is not often the case, provided manipulation of the cystoscope has been guarded and gentle, a tendency to tenesmus is shown, a warm anæsthetizing solution can be introduced through the second irrigating channel as soon as the last of the boric solution has issued from the first.

In most cases I prefer to get the urine through these short catheters and to personally hold the cystoscope carefully in place during the collection, although this is sometimes more arduous to the examiner than to the patient. When so gathered the resulting urines are pretty certain to be more reliable than if the telescope is drawn out over the catheters leaving the latter in the ureters. For, in such cases, even under favorable circumstances, a slight to and fro motion of the catheters attends withdrawal of the cystoscope, and the catheter tips are then liable to displace a litttle of the delicate ureteral epithelium, with a resulting exudation of some red blood cells, which then appear in the gathered urines. Such a return is not altogether satisfactory, even if one can pretty clearly ascribe this slight hæmaturia to some such trifling trauma inflicted by the catheter.

Moreover, the long catheters, whether the cystoscope is withdrawn or not, are apt to have their dangling ends exposed to some soiling contact and spoil any value which would otherwise attach to the urine cultures.

Notwithstanding the remarks just made about retaining the cystoscope in place and the employment in general of short catheters, I wish to take this occasion to express approval of Dr. Bayard Clark's suggestion to partially fenestrate the two catheter canals, so as to facilitate cystoscopic withdrawal when the catheters, whether short or long, are to be left in the ureters.

As this partial fenestration is made on the under side of the two canals (Fig. 8-A), and in no way jeopardizes the integrity of the separate irrigating channels or the guiding midrib between the two catheters, and at the same time retains all the original features of individual catheter support at the tip, the alteration in construction has been adopted.

Now that our collection is sufficient from both sides, one catheter after the other is slowly withdrawn from its ureter into the cystoscope. While doing so it is customary to turn on the light and watch each ureter mouth as the catheter leaves it. Then the telescope, with its catheters, is withdrawn from the sheath and the obturator again inserted before withdrawal of the sheath.

In exceptional cases it is well to use air as the distending medium for this work in place of water, but, since aqueous cleansing of the bladder is an almost invariable precursor to cystoscopy, it is much simpler to continue with this agent in the subsequent stages of the procedure than to bring into requisition the additional paraphernalia for air distention. The speaker's cystoscope, by reason of its independent sheath, which prevents the vesical lens from contact with the urethra during insertion, is equally well adapted for the employment of either medium. The cystoscopes which cannot well be used with air are those of the European pattern, in all of which a glass window or prismatic lens is an integral part of the shaft, and by contact with any mucous surface in transit becomes smeared so as to impair transparency. This haziness tends to clear up if the glass on entering the bladder meets an aqueous medium, but not if an aerial one.

When air is employed in cystoscopy it should enter the bladder at a temperature of at least 100 degrees Fahrenheit and by carefully graded pressure. The common method of inflation with the so-called air cystoscope, where a hand bulb is used to throw in air of the same temperature as that of the room, is always startling to the patient and quite generally painful. Of course, the impact of an air current gives a sense of cold in excess of the real temperature of the air, and it is well recognized that the liberation of compressed air is attended with the production of actual cold.

The least cumbersome and most practical way found by the speaker to overcome the defects of this crude method is by passing small jets of filtered air through a metal cylinder containing an electrically heated coil (Fig. 3). The hollow point of this cylinder is applied directly to the irrigating nozzle of the cystoscope, for if such coupling is effected by an intervening foot or more of rubber tubing the air will have lost a surprising amount of its heat. For feeding air to the heating cylinder a foot pump (Fig. 4) is advantageous, since it leaves the hands free for other work.

The heating cylinder should be suspended over the patient's left leg, for the same reason already given for placing here the irrigating hose and the electric cord to the cystoscope (Fig. 1).

Although for cystocopy, as explained, there is little occasion to employ air distension, it is surprising to note how rarely the services of this agent are appreciated or employed in suprapubic cystotomy, and how commonly the cumbersome and dirty rectal bag with water distension of the bladder are still adhered to.

For the experienced surgeon, peritoneal lifting by any form of bladder distension is commonly unnecessary, but when distension is resorted to at all in cystotomy air, properly used, has everything in its favor as compared with water. For such an operation and when the patient is under a general anæsthetic heating the air is superfluous.

Two years ago a well-known gynæcologist, when demonstrating his open-tube method of single ureter-catheterization on a female cadaver, surprised the speaker by saying: " Don't you wish this could be done in the male?" Interested as he evidently was in the subject, our work and brief papers must have escaped him, because in these it had been shown that already for two years we had been catheterizing male as well as female cases, and commonly both ureters at the same time, and by a method believed to be simpler than that he was showing for a single ureter.

The speaker may be pardoned if, influenced by an experience of this kind and from a source so little expected, he asks now to place on record the fact that his was the first cystocope devised for and successfully used to catheterize both ureters at the same time; then also pointing out that accurate deduction in regard to the functional capacities of the two kidneys could only be made when the excretions of both glands were gathered under exactly similar physiological conditions; in other words, synchronously, and with a catheter in each ureter.

In a paper entitled " What is the Best Method for Obtaining Urine Direct from the Ureters for Diagnostic Purposes?" read by invitation before the American Association of Genito-Urinary Surgeons in May, 1900,† the speaker, in alluding to the advantages he believed to be gained by his first double catheter cystoscope over others, which at this time were of the single-catheter type, stated that " In the future any device which does not provide for such synchronous collection as a routine measure must be considered antiquated."

This first double-catheter ureter cystoscope on the Brenner principle was devised by the speaker in 1898, and made for him by Leiter of Vienna. It was shown before the New York Surgical Society early in the following year, in connection with a paper entitled " The Differentiation of the Urines."‡ Wherein the speaker gave his reasons for advocating the direct rather than the prismatic or inverted image type of cystoscope for ureter-catheterization.

It is gratifying to note that the views then expressed have constantly been gaining adherents, particularly in this country, as well as that my latest instrument has been flattered by at least one imitation.

Although the original double-barreled ureter cystoscope was a definite step in advance, it had the fault of lacking irrigating facilities when the catheters were placed. Efforts to overcome this defect and yet not increase this cystoscope's circumference led to an altogether new plan in construction, described at the time as follows: " On finding it feasible to catheterize both ureters at the same time, and so get a more accurate expression of the separate renal conditions and functions than had attended the consecutive methods antedating it, we saw that an instrument, if of no greater circumference, which could admit of irrigation or evacuation of the distending medium would add materially to its value. After some experience, it was clear that, in order to economize sufficient space for the purpose desired, the terminal window, the setting of which took relatively a large area, must be discarded. By doing so we could utilize the two triangular crevasses between the telescope tube, the two catheter tubes and the sheath for irrigating channels. But without this window a hiatus was left at the heel of the cystoscope, rendering its introduction unsafe without an obturator, which must be withdrawn before giving place to the catheterizing telescope. Such a scheme at first appeared disadvantageous, but, as obstacles were overcome and new features added, we found ourselves possessed of a combination cystoscope where one common sheath performed the light-carrying and irrigating requirements for three different telescopic tubes. The sheath, once in the bladder, made interchange from one telescope to another painless and quick, while irrigation, with or without a telescope in the sheath, was facilitated. The method seems to open a new line of departure for cystoscopic advancement, in that a number of parts can be added to the present equipment, such as an application or operating telescope, a prismatic double-catheter telescope, or one which looks backward."

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†Medical and Surgical Report of the Presbyterian Hospital, vol. 5, page 70.

‡Annals of Surgery, Vol. XXX., p. 661.

The latest pattern of my bilateral catheterizing cystoscope (Fig. 5) aims at making this urine-collecting feature its main purpose, although the increased size of the visual field renders the catheter-telescope which goes with it a good one as well for direct general examination of the bladder; when so used rubber caps cover the empty catheter canals.

In this instrument the sheath is given an oval shape, so that the telescope may have a diameter equaling that of the two catheter canals.

Although not belonging to the set, a prismatic examining telescope can be ordered, but having to be cylindrical, in order to turn in the oval sheath, this telescope loses in size of visual field about as much as the catheterizing direct telescope gains. As in the first model, the irrigating channels are here given that otherwise waste triangular space existing bilaterally between the telescopic tube, the catheter canals and the inner face of the sheath.

The quality and arrangement of the lenses account in part for the clear visual field, while the noticeably good illumination—with a cold lamp—is due to a novel proximity of lens and lamp, which explains that prow-like protrusion of all the different telescopic tubes as shown in a profile view (Fig. 5), where the catheterizing telescope is so seen, and here is also to be seen that the underlying catheter canals extend well forward, (almost to the lens), for a double purpose, that of depressing an occasional fold of mucous membrane, which otherwise might partly occlude the lens. In a few cases, where the mucosa is quite œdmatous or very irregular, it has been advantageous to have the catheter canals extend forward a trifle beyond the lens, and so serve as even more of a depressor for the catheters as well as visual purpose.

The second object served by the extended and individual catheter canals is to give the flexible catheters sufficient support close up to the ureters in overcoming a resistance offered at times by the muscular fibres about their mouths.

If one needs an instrument which is primarily to be a large field examining cystoscope, both direct and prismatic and only secondarily for catheterizing purposes, the composite pattern (Fig. 6) is better. In this the sheath is cylindrical and the examining telescopes fit its lumen snugly, but the catheterizing telescope has here to be reduced to a size less than the diameter of the two catheter canals; still, it has served the requirements well since first introduced.

In this composite cystoscope, with cylindrical sheath, the latter is now fenestrated at its concavity, in order to permit the prismatic or retrograde telescope to look either through an aperture proximal to the lamp (Fig. 6B), or the same telescope, on being pushed an inch further in and a little to the other side of the lamp (Fig. 6C) acts as in the original model.

A non-hooded cold lamp was essential for the purposes of the projecting and revolving prismatic telescope, and as it proved equally serviceable for the direct telescopes, it is the only one now used with any of these instruments.

Although of different lengths and shapes (Fig. 9) the circumference of the sheaths of these varying cystoscopes is 24 Fr., unless specially ordered, when one of only 20 Fr. can be made (Fig. 8B).

In Figure 7 is shown different parts of an oval trocar and canula cystoscope, which form the basis for supra-pubic puncture examination. In this instrument, after the withdrawal of the trocar, a lamp, angled on its stem, is passed, followed by insertion of any of the three telescopes just described as a part of the seven-inch composite cystoscope. Only exceptional cases occur for its use, such as one of retention, requiring supra-pubic puncture, where the existing urethral, prostatic or bladder conditions will probably continue to render the ordinary methods of examination difficult if not impossible, and where the present and previous history leaves one in doubt as to the vesical and renal state. Here, so long as supra-pubic puncture is imperative, advantage may well be taken of it to view the bladder and to get sufficient urine from each kidney to enable the surgeon to determine the safest and most radical course of subsequent management. The separate parts of this supra-pubic cystoscope are removed in the reverse order of their introduction. Before withdrawing the canula a double-eyed soft rubber angled catheter of the Carlton pattern should be passed through it to the bas-fond, and the canula drawn out over the catheter.

The pierced bladder wall will have been stretched rather than cut by the trocar puncture, consequently the opening in it will hug the catheter tightly enough to prevent leakage. The bladder puncture should be preceded by a moderate scalpel incision of the parietes close to the symphysis, because, if the ureters are to be catheterized, the cystoscope should be as nearly vertical to their mouths as the pubic bone will permit, and in addition, this position for the drainage catheter is properly adjusted for the collapsed bladder.

RUPTURE OF BLADDER.

In Service of Dr. F. Tilden Brown.

John A., waiter, 32 years; single, Porto Rico.

Admitted March 8, 1904. Died March 8, 1904.

Diagnosis—Rupture of bladder, intra-peritoneal; peritonitis.

Habits—Markedly alcoholic.

Denies having had gonorrhea or syphilis.

Present history—On March 5, three days ago, in evening, while intoxicated he fell down flight of stairs, striking on sidewalk on outstretched arms, his abdomen coming in violent contact with pavement. He arose too dazed to walk and was carried home by friends. Went to bed but could not sleep on account of pain in lower abdomen. The next morning, because he had passed no urine and had persistent pain in his abdomen, went in an ambulance to a hospital, where he was catheterized and says that a large quantity of water was withdrawn. Walked home from hospital, went to bed suffering constant pain. Yesterday again went to the hospital by ambulance and again a large quantity of water was withdrawn by catheter. Returned home on foot and remained until this morning (March 8), when he took a street-car to Bellevue.

Since his injury there has been no voluntary urination or defecation, and he has taken no food because even water caused vomiting.

Physical examination—Rather corpulent man; heart and lungs negative. Abdomen distended symmetrically, tense, tender, marked pain on pressure, percussion tympanitic over central portion from pubic region to ensiform. Slight dullness in flanks but no wave of fluctuation felt. On admission was catheterized and forty ounces of bloody urine obtained. Fixed catheter in urethra and since catheterization twenty ounces have flowed out.

Temperature on admission, 99 degrees. One hour later, 101^2 degrees. Two hours later, 102^2 degrees. Pulse on admission, 136; has remained 136 since. Respirations have risen from 36 to 40 since admission.

Operation—Bladder test before operation 150 c. c., injected through catheter, and 150 c. c. returned. Catheter left in place.

Suprapubic laparotomy. Purulent fluid, intestines coated with plastic exudate, fresh adhesions binding coils together in region of the bladder and occluding it. Bladder exposed by freeing intestines adherent to it and a longitudinal tear 2½ inches long found in the postero-superior wall; catheter felt within the bladder through this tear. Fluid drained from perineal cavity and long rubber drainage tubes inserted into pelvis behind bladder. Condition of patient became very serious; hasty suture of wound and dressings applied. Patient died before removal to ward.

Autopsy showed the conditions in all organs which are found in chronic alcoholism, as well as rupture of the bladder and acute septic peritonitis. Culture from purulent peritonial fluid developed a diplococcus resembling the gonococcus.

Note—At so late a period after rupture and when the lesion has probably been occluded by adhesions (evidenced by measured injection test), laparotomy is counter indicated, and was here only justified by reason of the possibility of rupture of some other viscus and for peritoneal cleansing.

HYPERTROPHY OF PROSTATE—GALVANO-CAUTERY PROSTATOTOMY.

Service of Dr. F. Tilden Brown.

C. P. G.; age, 64 years. Admitted to Bellevue December 13, 1903. Complications, abscess of scrotum, cystitis.

Diagnosis—Hypertrophy of prostate. Complete retention of urine.

Family history—Negative. Parents lived to advanced age.

Personal history—Generous alcoholic habit. Fifteen years ago right inguinal hernia. Three years ago incomplete left inguinal hernia. Eight years ago, because of no favorable opportunity with a boating party, one day was passed without urinating; at night he was unable to do so as well as the following two days. When he was finally catheterized an immense quantity of urine was drawn. For two weeks he used a catheter himself and then had so painful a cystitis that he entered and remained in a hospital three months. On discharge the pain was relieved, but complete retention has always necessitated the use of catheter. Several times during his catheter life he has had painful swellings of the testicles. Ten days before admittance to Bellevue another such attack led him to seek hospital care.

On admission a large abscess of the right side of the scrotum existed with most marked fluctuations posteriorly. An indurated mass believed to be the epididymis led to a diagnosis of tuberculous epididymitis. Free incision, curettage and drainage was attended with prompt healing. A strepococcus only was recovered by culture and by microscopic examination of the pus and tissues.

During convalescence the cystitis was treated and the conditions causing retention studied. Rectal examination showed only a flat expansion of a but moderately enlarged prostate, apparently not sufficient to cause urinary embarrassment. A soft catheter was easily passed and the urinary distance was 9¼ inches. Cystoscopy showed a complete but slight collarette surrounding the internal meatus with a considerable bar at the base. The urine to the last drop was expelled with considerable force through the catheter. The abnormality was thought to offer admirable opportunities for correction and cure of symptoms by a galvano-cautery prostatotomy. This was done on February 13, 1904. The preliminary steps in the technique for Young's prostatectomy and Chetwood's prostatotomy being much alike an operator may, by being equipped with the instruments for both procedures, defer his choice until the finger has explored the bladder. Such a plan was pursued in this case, where the digital examination, through the perineal wound, verified the previous deductions drawn from cystoscopic, rectal and catheter examinations; namely, the existence of a collarette and bar, hence prostatotomy was proceeded with. A median and two lateral incisions were made with the cautery knife. A large perineal catheter was secured in place by sutures and retained for eight days. Convalescence was uninterrupted. At the end of one month the perineal fistula closed and all the urine was voluntarily voided through the urethra. The retrograde cystoscope showed the three hyperemic cicatricial furrows made by the cautery knife.

My own tests and those reported by others who have examined this patient concur in finding no residuum whatever.

Examination on June 1, 1904, reveals a clear urine, and reported to be passed only five or six times a day. No incontinence. Catheter passed after urination withdraws less than one-half drachm of urine. The patient has recently had a recurrence of abscess on the opposite side of scrotum, which healed promptly after incision and drainage. This patient dates his vesical disability from the time of a particularly careless inattention to a full bladder, but there is good reason to believe that for a considerable time antedating this occurrence he had been unconscious of incomplete evacuation, as is the case with many prostatics.

HYPERTROPHY OF PROSTATE—OCCASIONAL RETENTION, PERINEAL PROSTATECTOMY.

F. Tilden Brown, M. D.

J. V., stage manager; age, 53 years, Italy. Admitted to Bellevue Hospital March 4, 1904. Discharged May 5, 1904.

Family history—Negative.

Past history—Always has been well. When about eighteen years of age, through embarrassment, he once held his urine until he could scarcely pass it. This then became a habit and persisted for about ten years. He had good general health until about two years ago, when he went to a hospital with retention of urine, where he remained for a month. While there he was catheterized twice a day for the first three or four days; then he gradually recovered control of bladder and could pass urine fairly well.

Venereal history—None.

Present history—Eight days ago, after a period of constipation, he was unable to void urine and began to have severe pain in his bladder for which he entered Bellevue Hospital.

On admission—Complete retention; 26 ounces of urine were drawn; no blood. In the afternoon of this day 22 ounces were drawn. Patient occasionally passes some urine voluntarily and without pain. At other times he is unable to void any. This condition has required catheterization at some time during each 24 hours since admission.

Examination—Rectal reveals marked enlargement of each lateral lobe.

Cystoscopic examination shows very prominent lateral prostatic lobes; no median lobe. Chronic cystitis.

Operation—March 11, 1904., F. Tilden Brown, M. D.

Gas and ether. Extreme lithotomy position and with grooved staff in the urethra, an inverted Y incision is made in perineum, central tendon severed from the bulb and recto-urethralis muscle cut exposing the membranous urethra and capsule of the prostate. Young's prostatic tractor introduced to bladder through incision in the membranous urethra and the prostate drawn down into the wound. Incision made in each lateral lobe parallel with and outside the course of the ejaculatory ducts, and each lateral lobe, the size of a small egg, is removed separately from within capsule, leaving a bridge of prostatic tissue below the floor of the urethra in which the ejaculatory ducts are preserved uninjured. Perineal drainage tube inserted. Right half of wound closed. Sterile dressings.

Convalescence was uninterrupted. Tube removed March 17, sixth day. Out of bed March 20. When discharged from hospital, on May 5, the perineal wound was closed, the patient was voiding urine at normal intervals and the catheter test showed that there was no residuum.

The patient sent the following replies to questions* as to his condition under date of June 28, 1904: Wound is entirely healed; no pain in wound, bladder or urethra. Urination at intervals of two hours during the day and gets up once at night. Urinary control perfect. Has not been catheterized since operation. Sexual function "very good." General health very good; is attending to business. His only complaint is of slight burning sensation during micturition.

A test of the amount of residual urine eight months after operation showed this to be two drachms.

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*Murphy, Journal American Medical Association, May 28. 1904.

URINIFEROUS TUBULES IN THE URINE IN THE DIAGNOSIS OF RUPTURED KIDNEY.

Service of F. Tilden Brown.

P. C.; age, 38; driver, U. S.; admitted April 25, 1904; died May 11, 1904.

Diagnosis—Ruptured kidney, lobar pneumonia, fracture ninth and tenth ribs, sepsis.

History—In the morning of April 25, while driving truck, fell flat on pavement and fore wheel of truck passed over chest. He became unconscious and was later brought to Bellevue in ambulance.

Examination—Temperature, 99; pulse, 90; respiration, 24. Heart, negative; lungs, traumatic pleurisy, both sides of chest. Contusion right shoulder and inner side of left thigh, marked ecchymosis left thigh. Simple fracture of ninth and tenth left ribs. Tenderness left loin and over bladder. Passes considerable quantities of bloody urine. A measured amount of fluid injected through catheter into the bladder was at once recovered blood stained.

Cystoscopic examination—April 25, unsatisfactory. Field obscured by a single large blood clot of spherical form which occupied entire base of bladder. Rapid observation while irrigating with boric acid solution afforded partial view of normal bladder wall about this ball of blood clot, but catheterization of ureters could not be accomplished. Bladder was evidently intact. Tentative diagnosis made of rupture of one or both kidneys. Condition contra-indicated operation at this time.

The diagnosis of ruptured kidney indicated by physical signs and symptoms was made conclusive upon the first microscopic examination of the urine when numerous torn portions of uniferous tubules were seen in fields otherwise made up of blood cells, renal cells and various kinds of casts.

Temperature gradually rose, pulse and respiration increased. Consolidation in right lung was made out on April 28. Patient became progressively worse, delirious and cyanotic, with signs of fluid in both pleural cavities, and continued to live in this condition under stimulative treatment for two weeks.

AUTOPSY RECORD.

Well nourished and developed; rigor mortis present; discoloration left thigh and groin.

Pleural cavity, left side, adhesions between lung and pleura anteriorly and posteriorly.

Cavity filled with a bloody fluid; adhesions between pleura and pericardium.

Right side, adhesions above and below. Filled with a serosanguinous fluid.

Fracture ninth and tenth ribs on left side just posterior to angle.

Left lung, small in area, shows but slight crepitation. No crepitation in lower lobe; apex firm, some crepitation; slight congestion. Some oedema showing compression.

Right lung, markedly congested, lower lobe showing hypostatic pneumonia.

Liver, somewhat fatty.

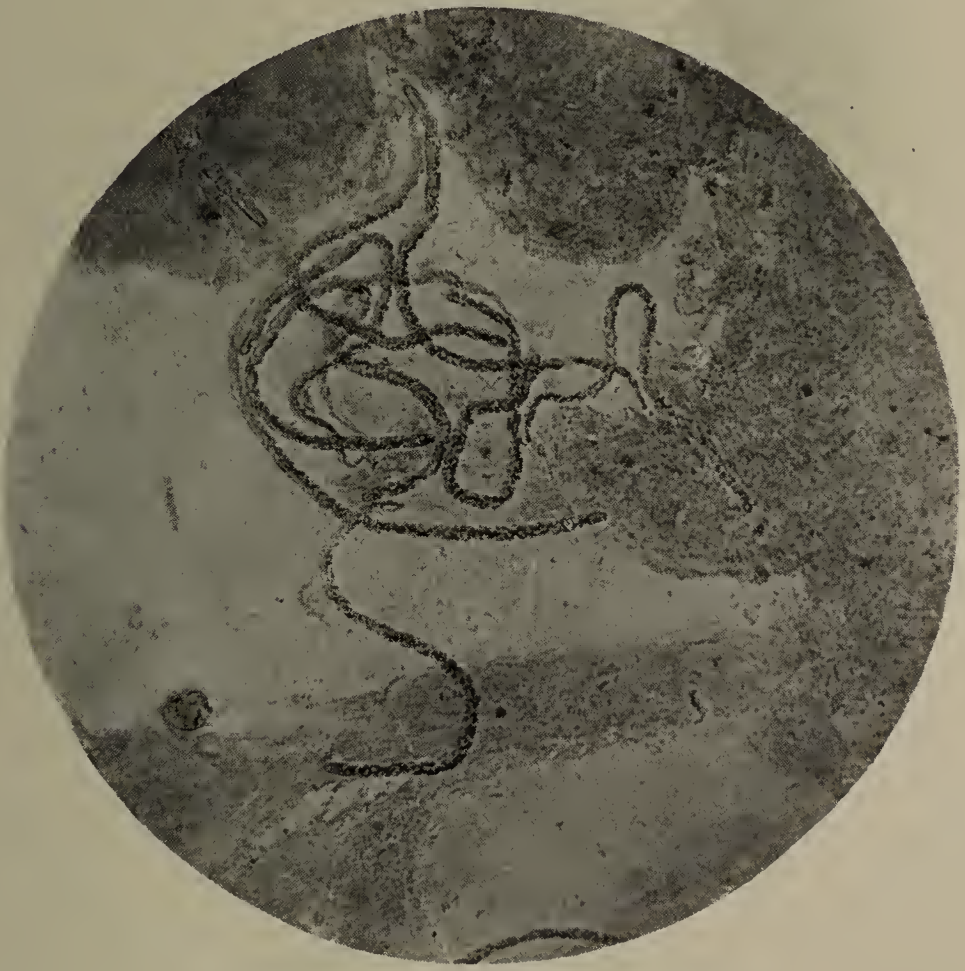
Spleen, very soft, friable, showing a marked septic condition; abscess on outer surface.

Left kidney, shows laceration in upper portion, septic infarcts, large gangrenous spots with abscess formations.

A CASE OF FILARIA BANCROFTI.

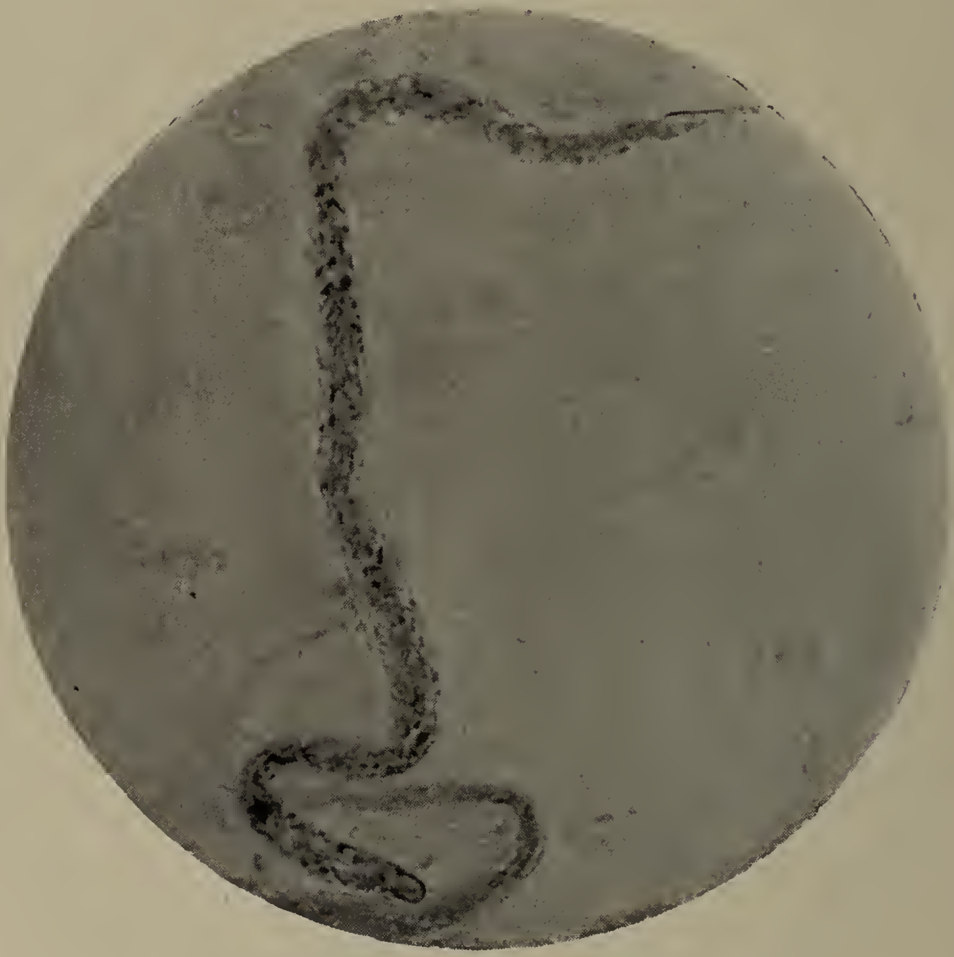
Service of F. Tilden Brown, M. D.

Associated with a mistake in clinical diagnosis, the writer had had the good fortune, on microscopical examination following operation, to find numerous embryos, in different developmental stages, of what he took to be Filaria Bancrofti. Although subsequent examination of the patient's blood had failed to show the embryos, he thought this might in part be explained by the supposition that the orchidectomy had removed all the adult female worms, if in fact more than one had been originally present. In such a circumstance, it was a question of some interest as to how



Embryos of Filaria sanguinis hominis.
Magnified 163.
The Writer's case : Scraped from a severed adult worm lying in a lymph space of the rete testis.

long the immature parasites of a possible earlier fecundation which had already been discharged into the circulation might be expected to persist. The history of the case in which these worms were so unexpectedly found substantiates what has lately been said by several writers regarding unusual parasitic infections, namely, that with the extension of the interests of this country in tropical lands we must be prepared for the manifestations of new diseases resembling perhaps quite closely those of diseases with which we have long been familiar. The patient, a German, thirty-four years old, who as a naturalized citizen had been in the United States Marine Service and stationed at Porto Rico for the past two years, had had no marked fever or illness of any kind. During the past two months he had first noticed an enlargement and slight tenderness of the left testis. The possibility of its being a barrier to re-enlistment led him to enter Bellevue Hospital for treatment. On examination, the writer found a moderate enlargement of the left testis. It was difficult to determine whether a slight but tense effusion in the tunica vaginalis was responsible for the symmetrical increase in size or not. A slightly



Embryo of Filaria sanguinis homines.
Magnified 410.
The Writer's case : Scraped from a severed adult worm lying in a lymph space of the rete testis.

tender nodule, the size of a very small oval marble, was felt where the mid-body of the left epididymis apposed the testis; which of these two parts was involved could not be determined. The lower part of the left spermatic cord was larger than its fellow. The left vas was also thicker, but not beaded. The patient had had no gonorrhea for ten years and had never had a swollen testis from this cause or from traumatism. A diagnosis was made of tuberculous epididymitis. The patient requested removal of the testis. At operation, nearly an ounce of the common straw-colored hydrocele fluid came from the left tunica vaginalis. This sac, together with the testicle, lower third of the cord, and considerable more of the vas, were removed. Examination of the specimen now showed that the small nodule occupied, not the epididymis, but the rete testis just underlying it. On cutting nearly through the lesion, a compact semi-fibrous perphery was noted, and in its centre a yellowish white substance somewhat firmer than, and only remotely resembling, tuberculous debris. The knife was drawn sidewise over this surface, and the small quantity of adherent material so scraped off the writer shortly afterward transferred to a couple of microscopic slides. On examining one of these stained for tubercle bacilli, some shriveled up worm-like forms were noted. The other slide was then treated with eosin glycerin and showed a number of scattered clusters of lumbricoids having the characteristics of Filariæ sanguinis hominis. These larvæ were distinctly granular and surrounded by the loose fitting hyaline membrane. What were taken to be very immature forms were of oval shape, and intermediate forms showed the embryo coiled and doubled in a larger sac, while those more advanced presented the elongated and undulating shapes seen when these parasites are found in the blood. The writer concluded that in cutting into the specimen he had opened a dilated lymph vessel or space, the abiding place of an adult female filaria; the knife had probably severed this worm, perhaps in several places, and on scraping it sidewise the uterine canals had been compressed and more or less emptied of their contents, in this way accounting for the great number of embryos found. The testicle and its attachments had unfortunately already gone to the pathological department before the writer had discovered its unexpected nature; by the time word was sent to have careful search made for the adult worm or worms the specimen had been considerably cut and placed in formalin solution. Many of the clinical features of this case were not unlike those met with and so thoroughly presented in the American Journal of the Medical Sciences for November, 1900, by Drs. Howard A. Lothrop and Joseph H. Pratt, of Boston. In their case they had the good fortune to recover seven female and two male Filari Bancrofti from the lymphatics of the epididymis.

While in a brother of their patient who had had no constitutional manifestations or local symptoms filaria embryos were found in the blood.

The excellent micro-photographs here reproduced of the writer's case are some of a number made by James H. Stebbins, Jr., Esq., and Dr. Frederic E. Sondern.

ROENTGEN RAYS AND STERILITY.

By F. Tilden Brown, M. D., and Alfred T. Osgood, M. D.,

New York.

Abrogation of that most ostensible function of the testes—spermatogenesis—among those who have worked a good deal in an X-ray atmosphere, is the altogether new, and, to some, alarming actuality found by the authors to have attended this occult influence. Not until data have been gathered from the great amount of material which should be available can even an approximate estimate be had as to what degree and kind of X-ray exposure the worker of average age and health must have experienced before azoöspermia ensues; and this, whether as a gradually progressive tendency or as a sudden climax after some particularly long and intense action of the rays.

The question now of greatest importance to many who find themselves affected with azoöspermia or oligo-necrospermia, is one of prognosis. It may be a matter of months or years before this can be answered.

It is reasonable to presume that, those, who for personal reasons have worked behind lead screens, or, for the conjoined interest of patient and self have used the so-called focussing tube-shields, will have escaped the consequences referred to.

The cases examined show that all of those who have been making X-ray work a specialty for the past three years—whether as medical men or artisans in electrical apparatus of this kind—are the subjects of total azoöspermia. A smaller number who have had much less exposure, but continuing up to the time of examination, show varying states of oligo-necrospermia.

A single example of sterility in a physician, who at the time of our discovery was being treated for oligo-necrospermia, with many misshapen forms, was found, on making fresh inquiries, to have had a good deal of X-ray exposure for one period of eighteen months, but for the past two years he had not been in the presence of an active tube.

Another case presented in his seminal fluid a seemingly normal number of active spermatozoa, and yet for a year or more he had been an occasional attendant at X-ray seances. This may point to the fact that the repeated prolonged exposures bring about the result.

In some 40 per cent. of the azoöspermia cases, none of whom had had any venereal disease or traumatism involving the genital tract, the interesting testimony was elicited that, having been married for some time before taking up the work, one or more children had been born, while since becoming X-rayists their wives have not been pregnant.

In view of Albers-Schoenberg's experiments upon rabbits and guinea pigs, it would seem that this result among Roentgen ray workers should not have been so surprising, when we realize that in much of their work they stand near enough to the patient to have the active rays strike the pelvic region of their bodies with considerable directness. The diagnoses in these cases have been based upon repeated careful examinations of seminal fluid obtained in reasonable quantity (grm. i.–iii.) by digital compression per rectum of the seminal vesicles, ampullæ of the vasa deferentia, ejaculatory ducts and the prostate, as well as (although not in all cases, unfortunately) of fluid obtained as a normal physiologic discharge.

None of the cases have been conscious of any deterioration or change in regard to potentia cœundi. The number of men examined thus far is sixteen, while reports substantiating these statements have been received from a number of others.

Dr. Lapowski reported a striking and interesting case before section on genito-urinary diseases of the New York Academy of Medicine on January 17, 1905, at which we made the first announcement of these findings. The case was one of fistula in ano with pruritus ani; advised against X-ray treatment. Examined before X-ray treatment; many active motile spermatozoa. Two exposures, ten minutes each. Examination showed oligonecrospermia. Two further similar exposures; examination showed azoöspermia. Examination four months later showed normal fluid, with many actively motile spermatozoa.

The insidious development of this condition without the slightest subjective warning leads us to call attention to the results recently reported by Halberstaedter* from a number of well-conducted experiments upon female rabbits which show the susceptibility of the ovary to this same influence to be even greater than that of the testis, and the result to be analogous.

We may also infer from his report that lead screens will serve to protect these organs from the injurious action of radiations from the X-ray tube.

The employment of Roentgen rays for fluoroscopic, skiagraphic and therapeutic purposes, in view of what we know of the injuries which may be produced by them, calls for the utmost care to protect all (operators, subjects and spectators) who may be exposed. All parts of the body not directly exposed for examination or treatment should be amply protected by means of efficient tube-shields, screens or metallic foil.

Appended is a list of some of the more important recent contributions to medical literature bearing upon our topic:

Albers, Schoenberg.....	Muenchener Medizinische Wochenschrift, 1903, No. XLIII., p. 1859.
Frieben.................	Muench. Med. Wochenschrift, 1903, No. LII., p. 2295.
Heineke................	Muench. Med. Wochenschrift, 1903, No. XLVIII., p. 2090.
Heineke................	Muench. Med. Wochenschrift, 1904, No. XVIII., p. 785.
Seldin..................	Fortschritte auf dem Gebiete der Rontgenstrahlen, 1903-1904, Dec. 9, Band VII., p. 322.
Baermann und Linser...	Muench. Med. Wochenschrift, 1904, No. XXIII., p. 996.
Halberstaedter..........	Berliner Klinische Wochenschrift, January 16, 1905, No. 3, p. 64.
Bergonie et Tribondeau.	Comptes rendu de la Société de Biologie, 1904, pp. 400-402 and pp. 592-596.
Philipp.................	Fortschritte a. d. Gebiete der Roentgen Strahlen, 1904, Band VIII., Heft 2.